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A Study of Maternal Deaths in the Province of Ontario*

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IT has been demonstrated that a very considerable proportion of the deaths from puerperal causes is preventable. With this fact in mind, a detailed study of maternal deaths in the Province of Ontario was instituted in 1933. At the outset it was clearly realized that mortality is not the only aspect from which this question might be investigated, but despite the limitations of this type of approach, it is the one which lends itself best to study.

In keeping with the trends in mortality and morbidity in other fields of public health effort, it would have been justifiable to expect a decline in the deaths from puerperal causes. Improved teaching of obstetrics in medical schools, the introduction of prenatal clinics in many centres designed to render possible supervision to those women who otherwise might find it difficult to obtain and educational efforts to stimulate the interest of women in seeking prenatal care early are some of the manifestations of effort in this field. In spite of these things, however, during the last 25 years puerperal mortality rates do not show any tendency to decline. Puerperal causes are still a factor in total mortality among women of child-bearing age second only to tuberculosis. For the five-year period 1927-1931, 17.7 per cent of all deaths among women 20-39 years of age were the direct result of pregnancy and child-bearing.

Figure I shows the trend of puerperal mortality in Ontario from 1909 to 1933. During this period maternal deaths were classified by cause in a fairly uniform way and records of maternal deaths may be considered reasonably reliable. It is evident from this figure that there has been no significant reduction in maternal mortality in Ontario during the last quarter of a century. Change

*Presented at the Twenty-Third Annual Meeting of the Canadian Public Health Association, Montreal, June, 1934.

†An abstract of this paper is published in the Canadian Medical Association Journal, 31: 655 (December), 1934, under the title, "Maternal Mortality in Ontario".

in the age distribution of mothers, an older age at marriage, or an increase in the proportion of primiparious women are all factors which influence the crude maternal mortality rate, but no excuse for the maintenance of existing puerperal death rates in Ontario can be found in such changes. For example, it is evident that the falling birth rate, while resulting in a greater proportion of primiparous births each year, also tends to smaller families and fewer women in the groups of higher orders of pregnancy. Since in both these groups the specific maternal risk is high, these changes tend to balance each other. Also, in the last decade, the trend in the distribution of confinements by age of mother in Ontario has actually been favourably to a lower puerperal death rate.

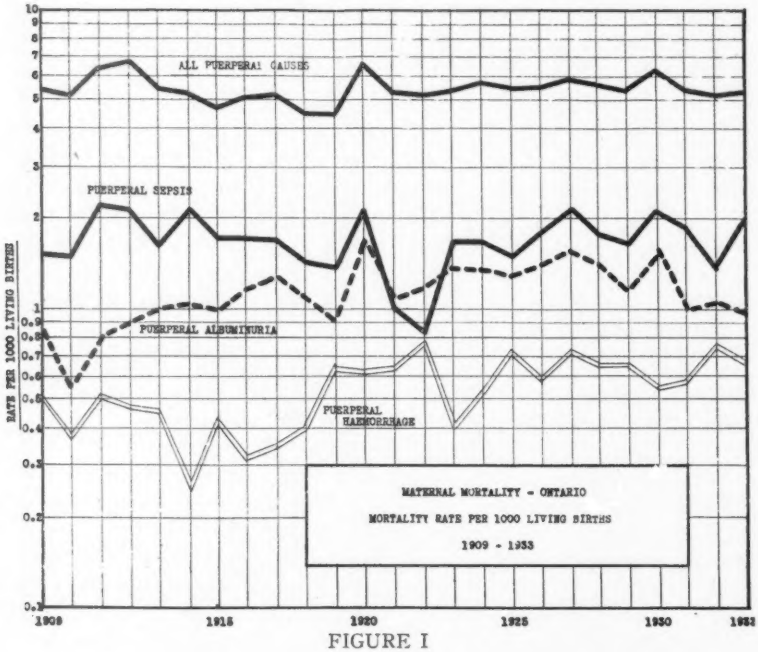

FIGURE I

It has been suggested that increasing completeness and accuracy of certification with greater public and professional interest in maternal deaths are partly responsible for the maintenance of puerperal mortality at its level of 25 years ago. Prior to 1909 it is likely that some of what we now call maternal deaths were missed in compilation, but in general during the last two decades only the probability of more accurate certification of septic deaths following abortion need be considered and we feel that the failure to reduce death from maternal causes is a real and not an apparent failure.

Figure I further shows the trend of specific mortality rates from puerperal sepsis (including septic abortions), puerperal albuminuria and puerperal haemorrhage for the same period. The mortality from puerperal sepsis

shows no decline, that from puerperal albuminuria and eclampsia shows a definite upward trend as a whole, though since 1920 the rate has never reached as high a point as it did in that year. The mortality from puerperal haemorrhage also shows a tendency to increase during the last twenty-five years.

SCOPE AND METHOD OF STUDY

In conducting this study the information ordinarily available in published vital statistical reports was supplemented by asking the attending physicians to supply further details concerning maternal deaths on an appropriate form. This information was sought in every instance in which pregnancy or a puerperal disease was recorded either as cause of death or as a contributory cause on the death certificate as received in the office of the Registrar General for the province beginning January, 1933. Separate forms were employed, differing only in minor points, the one for cases dying in hospitals, the other for cases dying in private homes. The former were collected by the Hospitals Division of the Ontario Department of Health through a statutory requirement and the latter by correspondence between the physicians in question and the Division of Maternal Welfare and Child Hygiene.

The detailed work of this study was undertaken in the Department of Epidemiology and Biometrics, School of Hygiene, University of Toronto. The coding of the deaths by cause was carried out in general according to accepted vital statistical practice which gives puerperal causes high preference over jointly-stated causes of death, particularly in the case of puerperal sepsis. When it was clear that a true puerperal cause existed, it was preferred to an extra-puerperal cause. When, however, a well-defined extra-puerperal entity was present and merely the existence of the puerperal state mentioned, the extra-puerperal disease was preferred.

The classification used throughout is that of the 1929 revision of the International List which effected (*a*) separation of abortions (with or without sepsis, 140 and 141) from puerperal sepsis proper (145), (*b*) specific mention of ectopic pregnancy (142), (*c*) sub-division of puerperal haemorrhage into placenta praevia and post partum haemorrhage (144 A and B), and (*d*) introduction of the new rubric no. 147, other toxaemias of pregnancy. In practice, rubric no. 147 was found to comprise only cases of pernicious vomiting and was therefore so named, while no. 150, other puerperal conditions, was called accidents of the puerperium. Rubric no. 143, other accidents of pregnancy (excluding haemorrhage), was not used, all the deaths being specifically classified to other rubrics.

For the year 1933 the number of puerperal deaths recorded by the Registrar General's Department was 334, and a total of 309 questionnaires was returned by physicians. In 257 of these, or over 75 per cent, the case referred to was a true puerperal death. In the other 52 cases it was judged that pregnancy was only an associated cause of death, the true cause being extra-puerperal in origin. To secure all available information concerning the remainder of the deaths for which no questionnaire was returned, a search of all the original death certificates for 1933 on file in the Provincial Registrar's Department was conducted, and records made of every case (other than those

for which questionnaires were already on hand) in which pregnancy or a specific puerperal cause of death was mentioned. This search yielded 77 true maternal deaths and 25 additional cases in which pregnancy was only an associated cause. In all then, there were 334 true maternal deaths and 77 deaths whose cause was extra-puerperal, but in which pregnancy was considered an associated factor. The details presented in the following pages refer (except where otherwise specified) to deaths in the former group, *i.e.*, to those which were considered true puerperal deaths.

MATERNAL DEATHS BY CAUSE

A summary of the deaths by cause is given in Table I.

TABLE I

TRUE MATERNAL DEATHS—ONTARIO, 1933

Cause (International List)	Number	Per cent
140—Septic abortion	48	14
141—Abortion without sepsis specified	11	3
(Specified as self-induced)	(21)	(6)
142—Ectopic gestation	11	3
144—Puerperal haemorrhage	43	13
145—Puerperal septicaemia	78	23
146—Puerperal albuminuria and eclampsia	62	19
147—Pernicious vomiting	6	2
148—Embolism, sudden death	40	12
149—Other accidents of labour (Caesarean section, dystocia, etc.)	29	9
150—Accidents of the puerperium	6	2
TOTAL	334	100

Puerperal sepsis (excluding septic abortion) was responsible for 23 per cent of the deaths. Almost 18 per cent were due to abortion, and of these more than one-third were said to be self-induced. Puerperal haemorrhage accounted for 13 per cent and puerperal albuminuria and eclampsia for 19 per cent of the deaths.

These facts demonstrate the significant part played by the four major causes, sepsis, toxaemia, abortion and haemorrhage, in the deaths studied. These four causes were responsible for three-quarters of all puerperal deaths during the year. The part played by sepsis and puerperal toxaemia deserves special emphasis since these deaths are largely preventable in character. The proportion of deaths ascribed to abortion serves to emphasize the very definite risk of pregnancy in the first trimester.

An observation of great import was that 27 of the 334 deaths, or over 8 per cent, were in single women, 18 (almost 70 per cent) of which were deaths from sepsis or abortion. The significance of this information lies in the fact that there is reason to believe the number of cases in which conception occurs before marriage to be high, particularly in young age-groups, and that specific puerperal risk is doubtless greater in such cases because these women do not ordinarily seek prenatal care and medical supervision early for obvious reasons.

VARIATIONS IN MORTALITY BY AGE AND CAUSE

The 334 deaths were distributed by age as follows: under 20 years, 3 per cent; 20-24 years, 21.6 per cent; 25-29 years, 22.4 per cent; 30-34 years, 22.7 per cent; 35-39 years, 21.6 per cent; and 40 years and over, 8.7 per cent. The relative importance of the various causes of death in each age-group is indicated in Table II.

TABLE II

DEATHS FROM SPECIFIED PUERPERAL CAUSES PER 100 DEATHS FROM ALL PUERPERAL CAUSES BY AGE ONTARIO, 1933

Cause	Age						
	Under 20	20–24	25–29	30–34	35–39	40–44	45–49
Abortions	10	21	19	18	17	11	0
Ectopic gestation	0	1	0	4	7	7	0
Puerperal haemorrhage	10	11	11	18	14	7	0
Puerperal sepsis	30	30	28	20	17	18	0
Puerperal toxaemias	20	17	24	14	25	22	50
Embolism and sudden death	20	10	9	16	10	15	50
Accidents of labour	10	7	9	7	10	15	0
Accidents of the puerperium	0	2	0	3	1	4	0
TOTAL	100	100	100	100	100	100	100

In the younger age-groups the part played by puerperal sepsis is greatest. In the older groups puerperal toxaemia is the principal puerperal cause, but deaths from puerperal toxaemias play a significant role in each age-group.

In order to eliminate variations due to chance which are inherent in using the figures for one year only, the proportion which each puerperal cause constituted of deaths from all puerperal causes in a given age-group was computed for the ten-year period 1921 to 1930. For the age-groups 30 and over, data refer only to the years 1923 to 1930 inclusive. Of necessity in this work the International Classification of Causes of Death of *1920* was employed. Table III presents the findings.

TABLE III

PER CENT DISTRIBUTION OF MATERNAL DEATHS BY CAUSE IN AGE-GROUPS ONTARIO, 1921 to 1930

Cause	15–19	20–24	25–29	30–34	35–39	40–44	45–49
Puerperal sepsis*	34.5	36.7	31.0	31.0	26.6	19.9	27.5
Puerperal albuminuria	31.1	26.9	22.5	20.8	24.5	23.4	17.5
Puerperal haemorrhage	8.1	7.5	8.5	9.9	14.0	17.4	12.5
Phleg. and embolism	2.1	6.2	7.6	10.0	10.2	12.0	2.5
Accidents of pregnancy†	9.4	9.5	13.4	11.0	10.2	7.6	20.0
Accidents of labour‡	10.2	7.7	11.7	13.9	10.9	16.8	20.0
Other puerperal causes	4.6	5.5	5.3	3.4	3.6	2.9	0
ALL CAUSES	100.0	100.0	100.0	100.0	100.0	100.0	100.0

*Including septic abortion.
†Including abortion (not septic) and ectopic gestation.
‡Including Caesarean section, difficult labour, rupture of uterus, instrumental delivery and uncontrollable vomiting.

It is apparent in this table that both puerperal sepsis (including septic

abortion) and puerperal albuminuria are relatively more important factors in the mortality among young women (15-24), while puerperal haemorrhage and embolism are relatively more important in the older age-groups. The increasing importance of puerperal haemorrhage with age is striking. Figure II illustrates these data.

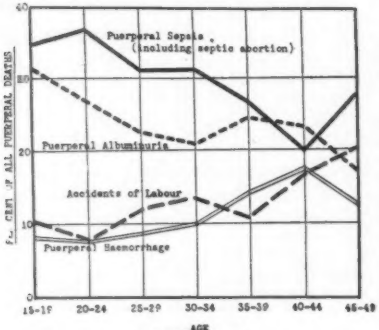

FIGURE II

Deaths from Certain Puerperal Causes Per Cent of all Puerperal Deaths by Age-Groups, Ontario, 1921 to 1930.

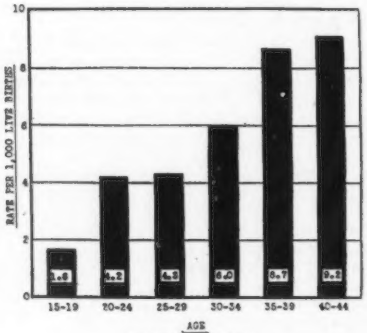

FIGURE III

Age Specific Puerperal Mortality Rates, Ontario, 1933.

To permit direct comparisons of mortality risk by age, specific death rates were calculated for each age-group for the particular data under study (Table IV).

TABLE IV

AGE SPECIFIC PUERPERAL MORTALITY RATES†
ONTARIO, 1933

Age	Live Births		Maternal Deaths		Rate per 1,000 live births
	*Per cent Distribution, 1932	Estimated for 1933	Number	Per cent	
Under 15	0.02	15	2	0.6	...
15–19	7.83	4,992	8	2.4	1.6
20–24	26.72	16,993	72	21.6	4.2
25–29	27.21	17,305	75	22.4	4.3
30–34	20.06	12,758	76	22.7	6.0
35–39	13.08	8,318	72	21.6	8.7
40–44	21.61	2,932	27	8.1	9.2
45–49	0.45	286	2	0.6	...
TOTAL	100.0	63,597	334	100.0	5.3

†Based on distribution of live births in 1932 by age of mother.
*Omitting those in which age was not stated.

The figures show that in this particular year the specific mortality rate was lowest among women 15-19 years of age. The rates increase with age throughout, the three age-groups under 30 years all having rates below that for all ages, while in the three age-groups 30 years and over, the mortality is very much higher than that for all ages combined (Figure III). It is surprising to find such a low rate in the age-group 15-19 years and only once before in Ontario in the last 25 years has this occurred. Since the number of deaths recorded among women at these ages is small, an annual rate is subject to

considerable chance variation. It seems probable, however, that the death rate in the youngest group (15-19 years) *may actually be lower* than that in other age-groups and that the excess risk which has been demonstrated in this group is due to factors other than age *per se*. The chief of these are two variations in the composition of those women at risk in this age-group which have been shown to influence mortality strikingly, (1) a much greater proportion of unmarried mothers and (2) a higher proportion of primiparae. These variations both tend to increase the apparent risk at the younger ages apart from the influence of age alone. As a whole, the evidence provided in this study clearly demonstrates the increasing risk in pregnancy and childbearing with increasing age of mother, and suggests that the age specific risk may actually be lowest among women 15-19 years, all other things being equal.

RELATION OF MATERNAL DEATHS TO OUTCOME OF PREGNANCY

The maternal deaths were classified according to whether the outcome of the pregnancy was a living birth, a stillbirth or "no birth", the latter including those cases in which death occurred before the onset of labour and cases in which the foetus was under the age of viability. The data are presented in Table V. Those cases in which this information was not available are omitted in computing the percentages in the respective groups.

TABLE V

PUERPERAL DEATHS BY CAUSE SHOWING OUTCOME OF PREGNANCY
ONTARIO, 1933

Group	Cause of Death (International List Number)								Total	Per cent
	140, 141 and 142	144	145	146	147	148	149	150		
Living birth	0	22	56	27	0	24	18	4	151	47
Stillbirth	0	15	15	17	0	10	10	1	68	21
No birth*	70	4	1	18	6	4	1	0	104	32
No data	0	2	6	0	0	2	0	1	11	..
TOTAL	70	43	78	62	6	40	29	6	334	100

*Includes the cases in which the foetus was not of viable age or cases in which woman died undelivered of a viable pregnancy.

Forty-seven per cent of the maternal deaths were associated with a live birth, 21 per cent with a stillbirth and in 32 per cent the fatal outcome occurred either before delivery or before the period of viability was reached. This again emphasizes the fact that mortality in the first two trimesters is a very definite concern in considering a solution of the public health problem presented by maternal deaths.

It is of interest, too, in this connection, that only in about half of the delivered cases was the pregnancy full-term.

ORDER OF PREGNANCY

The risk of death in pregnancies of various orders is of great interest in an approach to this problem. Of the maternal deaths during 1933, 33 per cent

were in primiparae, while 28.5 per cent of legitimate births (and probably all illegitimate births) belonged to the group of first order pregnancies. To ascertain the risk according to number of pregnancies, the deaths were grouped according to the order of the pregnancy and specific mortality rates were calculated for each group based on the number of live births (estimated) in that particular group. This was done by assuming that the distribution of legitimate births by "order of birth" for 1933 was not significantly different from that in 1931, the per cent distribution in the latter year by order of birth being applied to the total births for 1933 to obtain the figures in column 2 of Table V. Since practically all illegitimate births are first order births, they were added to the estimated legitimate births in the primiparious group. The number of deaths in which order of pregnancy was not obtainable was unfortunately rather large and these were redistributed arithmetically. (This procedure is of course open to question.)

TABLE VI

SPECIFIC MATERNAL DEATH RATE BY ORDER OF PREGNANCY
ONTARIO, 1933

Order of Pregnancy	Estimated Live births*	Deaths† No.	Rate
1	20,108	109	5.4
2	13,550	62	4.6
3	9,147	47	5.1
4	6,379	29	4.5
5	4,379	19	4.3
6	3,095	19	6.1
7	2,220	16	7.2
8*	4,719	33	7.0
TOTAL	63,597	334	5.3

*1st pregnancy group includes 2,782 illegitimate births.

†62 cases in which order of pregnancy not obtainable redistributed proportionately.

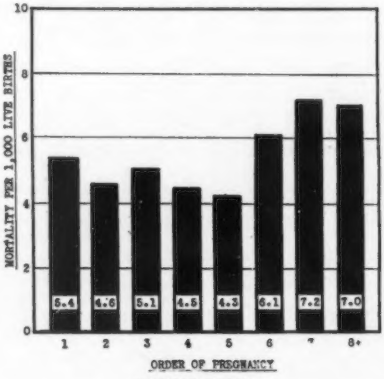

FIGURE IV

Specific Puerperal Mortality Rates by Order of Pregnancy, Ontario, 1933.

On this basis the risk appears to be lower in the second to fifth pregnancies than in the first or succeeding ones, but pregnancies of higher order than the fifth appear to carry a risk greater than the first one. Figure IV illustrates these variations. As a whole the risk in primiparae is 5.4 and in multiparae 5.2 on this basis.

VARIATION IN MORTALITY BY PLACE OF RESIDENCE

The reason for many of the differences in urban and rural mortality is shrouded in doubt. Unfortunately re-allocation of births and deaths by place of residence is not generally undertaken as in England and comparison of urban and rural mortality experience in Canada is, therefore, ordinarily unjustifiable. It was possible in this study, however, to allocate all deaths by place of residence. Four groups were defined: (1) rural, (2) cities of 10,000 and over, (3) towns of 5,000 to 10,000, and (4) towns and villages of 1,000 to 5,000 (1931 census). The live births excluding non-residents for groups (2) and (3) were estimated for 1933 from those for 1932. No resident figures

for group (4) were available, but this would not affect the relative result in any case. On this basis, the rates for the 4 groups were, respectively, 4.7, 6.2, 3.6 and 4.2. The high rate of 6.2 in cities of 10,000 and over compares unfavourably with the low rate of 4.7 in rural areas. This unfavourable comparison must be considered in the light of the following facts: (1) birth rates are lower in urban centres and there is, therefore, a relatively higher proportion of primiparae exposed at a higher puerperal risk; (2) hospitalization and operative interference is very probably more frequent in urban communities; (3) intangible differences in the apparent risk among urban as compared with rural mothers on the score of the "personal equation".

The causes responsible for the urban and rural variations are shown in Table VII, which gives specific mortality rates by cause for the rural group and the three "urban" groups combined.

TABLE VII

SPECIFIC MORTALITY RATES BY PLACE OF RESIDENCE AND CAUSE
Ontario, 1933

Place of Residence	Live Births Estimated	Cause of Death														All Causes	
		Abortion		Haemorr.		Sepsis		Toxaemia		Embolism		Others†					
		No.	Rate	No.	Rate	No.	Rate	No.	Rate	No.	Rate	No.	Rate	No.	Rate		
Rural....	29,204	19	0.6	21	0.7	30	1.0	31	1.1	19	0.6	18	0.6	138	4.7		
Urban*	34,393	40	1.2	22	0.6	48	1.4	37	1.1	21	0.6	28	0.8	196	5.7		
Total....	63,597	59	0.9	43	0.7	78	1.2	68	1.1	40	0.6	46	0.8	334	5.3		

*Including cities and towns of 1,000 and over. Correction for residence made on basis of 1932 returns, except for villages of 1,000-5,000, and for Forest Hill and Swansea.
†Including ectopic gestation, accidents of the puerperium and others.

Practically all the difference in mortality between the two groups is due to differences in mortality from puerperal sepsis and abortion. The rate from abortion in "urban" areas is twice that in rural parts and the death rate from puerperal sepsis (excluding abortions) is 40 per cent higher in urban than in rural areas. This problem requires further careful study.

TYPE OF DELIVERY

Of the 257 maternal deaths for which completed questionnaires were returned, 192 were delivered of live or stillborn infants. In 103 of these delivered cases, or 54 per cent, delivery was non-spontaneous (induced, instrumental or operative) in character. It was noteworthy that in this series there was no appreciable difference between the type of delivery among urban and rural mothers. Of 60 delivered cases dying of puerperal sepsis, 40 were cases in which there was no record of any interference.

To afford a reasonably specific comparison of mortality by type of delivery, it was assumed that all of the remaining 29 delivered cases for which no questionnaires were available (except one in which a Caesarean section was done) were spontaneous deliveries. In all, then, there were 104 non-spontaneous and 117 spontaneous cases. An attempt was made to estimate the number of deliveries which were "operative" and spontaneous, respectively.

To do this twenty per cent of all live births were considered to follow operative delivery (New York City estimate). This estimate is probably high, so that both this procedure and that of accepting all the delivered cases for which no details were available as spontaneous, favour a lower rate in the operative group. On this basis the *mortality rate in the non-spontaneous group was 8.2 per 1,000 live births and 2.3 in the spontaneous group.*

HOSPITALIZATION

In a study of this sort it would be of practical interest to know some details of the hospitalization of the cases. How many were hospitalized by previous arrangement? How many were taken to hospital as an emergency measure?

It was found that 61 per cent of the delivered cases were delivered in hospital and 39 per cent at home. In contrast to this, 73 per cent of the deaths occurred in hospital and 27 per cent at home. Hence at least 12 per cent of the fatal delivered cases were removed to hospital because of some complication or *in extremis.*

A further attempt was made to secure a comparison of institutional and domiciliary rates for delivered cases. Deaths from abortion and ectopic pregnancy, as well as other deaths before delivery, were excluded. A case was then called institutional if the woman was delivered in hospital and died there, and domiciliary if she was confined at home and died either at home or in hospital. Total deaths and deaths from each cause in two groups—institutional and domiciliary—are given in Table VIII. Specific mortality rates were computed for each group respectively, using estimated live births for "institutional" and "domiciliary" groups based on those recorded in the Registrar General's report for Ontario, 1932.

TABLE VIII

SPECIFIC DOMICILIARY AND HOSPITAL MORTALITY RATES BY CAUSE*
ONTARIO, 1933

Cause of death†	Institutional		Non-Institutional	
	Number	Rate per 1,000 live births	Number	Rate per 1,000 live births
Puerperal haemorrhage	21	0.8	17	0.4
Puerperal sepsis	41	1.7	28	0.7
Puerperal albuminuria	32	1.3	13	0.3
Embolism, sudden death	12	0.5	22	0.6
Accidents of labour	22	0.9	7	0.2
Accidents of the puerperium	3	0.1	3	0.1
TOTAL	131	5.3	90	2.3

*Based on institutional and non-institutional live births estimated for 1933 from 1932 figures.

†Delivered cases only.

Estimated institutional live births (39 per cent)..... 24,800
Estimated non-institutional live births (61 per cent)... 38,800

In all cases except embolism and accidents of the puerperium, the low domiciliary rates are striking. The much higher rate from puerperal albuminuria in institutional cases is doubtless related to the fact that these women went to hospital for treatment before delivery and a number of them had

Caesarean sections done for this condition. The higher mortality from puerperal haemorrhage and accidents of labour is also to be expected. The higher rate from puerperal sepsis in the institutional group, however, is not so easily explained.

These comparisons may be criticized on the ground that only those deliveries which appear likely to be easy and normal will be undertaken at home. It would certainly seem, however, that the differences are too great to be accounted for on these scores alone.

CAESAREAN SECTION

Operative interference in the form of Caesarean section is becoming more frequent. Hawkes has shown that the incidence of Caesarean section in the New York Nursery and Child's Hospital rose from 2 per 1,000 deliveries in 1910 to 25 per 1,000 deliveries in 1927. In this study it was found that in 40 of the 334 deaths, or 12 per cent of all maternal deaths during the year, a Caesarean section had been performed. Of delivered cases only, the proportion in which a Caesarean section was done was approximately 18 per cent. For those 257 cases for which questionnaires were completed, it was found that of those delivered, among primigravidae, 28 per cent were delivered by Caesarean section and among multigravidae, 16 per cent.

The indications given for operation were as follows: eclampsia in 13, contracted pelvis in 5, disproportion in 3, inertia and prolonged labour in 4, dystocia in 3, previous Caesarean sections in 2, malpresentation in 2, accidental haemorrhage in 3, ruptured uterus in 1, cardiac disease in 1, previous accident in 1, and indications not evident in 2.

The deaths in which a Caesarean section was performed were distributed according to place of residence, four divisions being made as before. In order roughly to compare the groups, the rates per 1,000 live births were computed as in the following table, using the distribution of live births during the previous year as a basis.

TABLE IX

CAESAREAN SECTION DEATHS BY PLACE OF RESIDENCE
ONTARIO, 1933

Place of Residence	Caesarean section		Resident live births (1932)		Est'd births 1933	Rate per 1,000 live births
	Number	Per cent	Number	Per cent		
Cities (10,000 and over)	23	57	27,255	40.77	25,928	0.9
Towns (5,000-10,000)*	3	8	3,196	4.78	3,040	1.0
Towns and villages† (1,000-5,000)	4	10	5,701	8.53	5,425	0.7
Rural‡	10	25	30,690	45.92	29,204	0.3
TOTAL	40	100	66,842	100.00	63,597	0.6

*No correction made for residence in case of Forest Hill and Swansea.
†Non-residents not excluded.
‡Including villages of less than 1,000 population.

These data show that the incidence of Caesarean section is more than twice as high among mothers of urban than among those of rural areas.

PRENATAL CARE

Much has been said as to the value of medical supervision during the antenatal period. It is not possible even to guess how frequently or how adequately such service is rendered to pregnant women in general. It seems reasonable to suppose that the percentage of those dying who maintain a reasonable contact with their physicians during the last two trimesters is as great as the percentage among all pregnant women. In order that accurate information on this point may be obtained, the authors suggest that the available space on the physician's notification of birth be used for questions re (1) prenatal care in the mother concerned and (2) type of delivery. Even, however, if the proportion having had prenatal care in those dying *is* as great as in pregnant women as a whole, the fact still remains that *many of the deaths from certain puerperal causes could be prevented if prenatal care were secured in all cases.*

The data asked in connection with prenatal care were (1) date on which the patient first consulted her physician and (2) what prenatal care, treatment or instruction did the patient have between that date and her admission to hospital. The replies to these questions were frequently incomplete so that there was no sound basis for a rigid classification of prenatal care in a given case. The classification used was "adequate," "inadequate" and "none". A case was judged to have had adequate prenatal care if the woman had had what seemed to be supervision adequate to her protection. If it was doubtful whether the supervision was adequate, or inadequate, it was called adequate. None meant no supervision whatever.

The findings are summarized in the next table, based on data from the 257 questionnaires returned (excluding abortions and ectopic pregnancies).

TABLE X

SUMMARY OF PRENATAL CARE BY CAUSE*
ONTARIO, 1933

Cause of Death	Adequate		Inadequate		None		No Data	Non Co-op	Total
	No.	Per cent	No.	Per cent	No.	Per cent			
Puerperal haemorrhage	13	41	6	18	13	41	5	(3)	37
Puerperal septicaemia	24	47	10	20	17	33	10	(3)	61
Puerperal toxaemias†	17	28	22	35	23	37	3	(4)	65
Embolism and sudden death	9	37	9	37	6	25	4	(5)	28
Other accidents of labour and accidents of the puerperium	17	71	3	12	5	17	5	(2)	30
TOTAL	80	41	50	26	64	33	27	(17)	221

*Percentages exclude those "not stated."
†Includes pernicious vomiting.

Thus 41 per cent were described as "adequate," 26 per cent as "inadequate" and 33 per cent as "none". It is of special significance, in the light of what we know regarding the value of prenatal care in relation to puerperal toxaemia, that only 28 per cent of those dying from this cause received adequate supervision and 37 per cent received none. Table XI shows the variations in prenatal care by place of residence.

TABLE XI

PRENATAL CARE BY PLACE OF RESIDENCE*
ONTARIO, 1933

Residence	Adequate		Inadequate		None		Total
	No.	Per cent	No.	Per cent	No.	Per cent	
"Urban"†	53	50	28	26	26	24	107
Rural	27	31	22	25	38	44	87
TOTAL	80	41	50	26	64	33	194

†Includes cities, towns and villages of 1,000 and over.
*Excluding abortions and ectopic pregnancies and omitting cases in which no information was given.
(These figures are slightly different from those appearing in the preliminary report in Canad. M. A. J., Dec., 1934.)

Sixty-nine per cent of the women dying in rural areas had inadequate or no prenatal care in contrast to 50 per cent in urban centres. This is to be expected since it is only in the large centres that any provision is made for prenatal supervision for those whose financial status precludes seeking medical care before delivery.

DEATHS FROM EXTRA-PUERPERAL CAUSES

The number of deaths in which pregnancy was only considered a contributory factor numbered seventy-seven. These were divided into 2 groups: I, those in which a disease existed before pregnancy (pulmonary tuberculosis, heart diseases, etc.); that is, those women to whom pregnancy was an especial risk because of a pre-existing disease, and II, those in whom a fatal disease developed during pregnancy. Thirty-three of those dying from extra-puerperal causes were in the first group and 44 in the second. Table XII gives the causes of death in these two groups.

TABLE XII

EXTRA-PUERPERAL DEATHS BY CAUSE

GROUP I		GROUP II	
Disease Causing Death	Number	Disease Causing Death	Number
Pulmonary tuberculosis	4	Scarlet fever	3
Carcinoma of rectum	1	Influenza	13
Carcinoma of uterus	1	Mastoiditis	1
Carcinoma of breast	2	Coronary embolism	1
Rheumatic fever	2	Phlebitis (varicose veins)	1
Diabetes mellitus	1	Bronchopneumonia	1
Diabetes insipidus	1	Lobar pneumonia	10
Pernicious anaemia	5	Pneumonia (not specified)	8
Leukaemia	2	Appendicitis	2
Banti's disease	1	Acute yellow atrophy of liver	3
Cardiac disease	10	Cholecystitis	1
Chronic nephritis	3		
TOTAL	33	TOTAL	44

In general, cardiac disease, pulmonary tuberculosis and "pernicious" anaemia were the most common disease in the first group, the pneumonias and influenza in the second. In group I, it was found that the proportion of stillbirths was higher than was the case among puerperal deaths as a whole.

PHYSICAL CONDITION BEFORE PREGNANCY

Of the 257 maternal deaths for which questionnaires were available, in 202 cases an answer to the query "physical condition before becoming pregnant" was given. The replies were classified thus: "good", "fair", "poor", and "very poor". On this basis, 34 or 17 per cent of the replies stated that the state of health before pregnancy was "poor" or "very poor". While it should be kept in mind that we have no control data from an unselected group of women who survived pregnancy and childbearing as to their physical condition before pregnancy, it is reasonable to suppose that the proportion among them in poor or very poor health would not be as high as one in six.

MEDICAL CARE

Skill and judgment on the part of the medical attendant are essential factors in a favourable outcome of pregnancy and labour. These things are requisite too for the subsequent welfare of both mother and infant. A careful study of the 257 questionnaires from this point of view, however, showed that it was impossible to assay the quality of medical care from such a source. An analysis was therefore made of the cases according to the time at which medical care was received in relation to labour, terminal illness or death.

(*a*) *Abortions.* As pointed out in Table I, 59 or 17.7 per cent of all maternal deaths during the year were due to abortion, and of these 21, or more than one-third, were specified as self-induced. Thirteen of these 59 deaths were in unmarried women. Questionnaires were available for only 26. Ten among this group were seen by a physician only after sepsis had developed or when *in extremis*. Two were seen only after death. In 2 cases a curettage was performed on a septic patient.

(*b*) *Ectopic Pregnancy.* Of 11 deaths from this cause, details were available for 10. Three were seen before rupture of the tube occurred and 6 after rupture or severe haemorrhage. One case was moribund when seen. Of these 10 cases, 6 were operated upon and the tube removed, and in 3 of these latter cases it was recorded that a transfusion was given. Two patients died before an operation could be performed. In the other 2 a ruptured ectopic pregnancy was discovered at autopsy.

(*c*) *Puerperal Haemorrhage.* The problem of haemorrhage in pregnancy and childbirth is of extreme importance. It has been recently stated that at least one-half of the deaths from haemorrhage could be prevented if both expectant mothers and doctors realized the danger and took proper steps to control it. The work of Aschheim, Zondek and others has established the concept that any uterine haemorrhage occurring during pregnancy may be a source of danger to both the life of mother and foetus.

Physicians' replies were available respecting 37 of the deaths from puerperal haemorrhage. In 8 cases the patient was seen by a physician only after severe haemorrhage or *in extremis*. One case (attended by a midwife) was seen by a physician only after death. In 5 of the 16 cases of placenta praevia, it was stated that a transfusion was given. In 2 cases warning haemorrhages had not been heeded by the patient. In 2 cases manual

removal of a retained placenta was done. Three women died undelivered of viable pregnancies.

Of 21 cases of post partum haemorrhage, transfusion was mentioned as treatment in 3 cases. In 1 case manual removal of placenta was performed and in 2 others the placenta was adherent or partially separated and not expelled. All cases were delivered.

(*d*) *Puerperal Septicaemia.* Of 78 deaths from puerperal septicaemia, details on supplementary forms were returned for 61. The following table gives a brief analysis of medical care in this group.

TABLE XIII

DEATHS FROM PUERPERAL SEPTICAEMIA

	Number
Physician in attendance before labour	38
Physician called first during labour	15
No physician at the labour	3
Not sufficient data	5
TOTAL	61

Of those in which a physician was in attendance before labour, 11 or about one-third were in "poor health" when seen.

The following additional facts were evident on further study. There were 5 cases of retained placenta. In 3 of these it was delivered manually (in 1 of these it was first discovered 4 weeks after delivery, and the patient died 24 hours after the removal). In another case the retained placenta was discovered at autopsy. In the other case, there was a small retained accessory placenta which was expressed on the third day post partum. In 2 of these fatal septic cases the foetus had been dead several days before delivery. In 6 cases a positive blood culture was obtained and in 3 cases (not the ones in which positive culture was mentioned) scarlet fever antitoxin was given. In 1 case it was stated that the patient "did not observe the physician's orders re sterile dressing". Only 1 case was attended at labour by a midwife. Of 29 cases in which the presentation was stated, in 8 it was specified as breech. In 7 cases death from sepsis followed Caesarean section.

The onset of illness in relation to labour in 51 of the cases was given as follows:

TABLE XIV

DEATHS FROM PUERPERAL SEPSIS

Onset of Illness	Number
Before labour	4
Immediately post partum	8
1 day post partum	8
2 days post partum	9
3 days post partum	8
4 days post partum	7
5 days post partum	2
6 days post partum or later	5
TOTAL	51

(*e*) *Puerperal Albuminuria and Convulsions.* Of 62 maternal deaths in this group data were on hand for 59 of them. Table XV presents the findings in this group:

TABLE XV

DEATHS FROM PUERPERAL ALBUMINURIA

	Number
Physician in attendance before terminal illness	35
Physician in attendance at terminal illness only	8
Physician called when patient was in convulsions	8
Physician called after labour	1
Physician called after death	1
Not sufficient data	6
TOTAL	59

Of the group of 35 which were attended by physicians before labour, 12, or 30 per cent, were "sick" when first seen by the doctor. In 4 cases the patient could be said to be non-cooperative.

Of the 59 cases 17 died undelivered and of the remaining 42 delivered cases, 12, or *30 per cent were delivered by Caesarean section.* In 4 of these latter the patient was in coma or having convulsions.

(*f*) *Pernicious Vomiting.* Six cases are recorded. In 3 a therapeutic abortion and in 1 a curettage was done. In 2 cases there had been no prenatal supervision at all. Period of gestation ranged from 8 to 20 weeks.

(*g*) *Embolism and Sudden Death.* Of 40 deaths allocated to this group of causes details were available in 28. Of these, 17 were cases of pulmonary embolism. In 8 other cases other causes were preferred to embolism, so that in all there were actually at least 25 cases in which pulmonary embolism occurred.

Of the 28 cases, 26 were delivered, and in 7 of these the presentation was breech, face or transverse. In 1 case the placenta was retained. Five cases could be described as non-cooperative, 3 of whom declined hospitalization when it was advised. In 5 cases the patient was first seen by the physician when in labour. In 1 case (attended by a midwife) there was no physician in attendance at the labour.

(*h*) *Other Accidents of Labour* and Accidents of the Puerperium.* In this group there were 35 cases, details being available in 30. Two cases of puerperal insanity are included. In 4 cases physician was called in labour and 1 case was not attended at labour by a physician (midwife case). There were 5 cases of ruptured uterus in this group of cases, 2 of them in women who had had previous Caesarean sections. In another of them, pituitrin was given before delivery.

*Includes Caesarean section, dystocia, instrumental delivery, rupture of uterus and other accidents of labour.

SUMMARY

1. There has been no significant reduction in mortality from puerperal causes during the last twenty-five years.

2. Mortality due to pregnancy and childbirth is second only to tuberculosis in women of childbearing age. Among women 20-39 years of age, 17.7 per cent of all deaths during the five years 1927-1931 were puerperal in character.

3. During 1933 there were 334 true puerperal deaths in Ontario and 77 others in which pregnancy was an associated cause. The puerperal death rate for the year was 5.3 per 1,000 live births.

4. Data were collected through special questionnaires and replies were received for about 75 per cent of the cases.

5. The per cent distribution of the deaths by cause was as follows: puerperal septicaemia 23, puerperal toxaemia 20, abortion 18, haemorrhage 13, embolism and sudden death 12, and other puerperal causes 14 per cent.

6. Eight per cent of the deaths were in single women, 70 per cent of which were due to puerperal sepsis or abortion.

7. Age is shown to be a striking factor in deaths from puerperal haemorrhage, mortality rising as age increases.

8. Age specific mortality rates demonstrate an increasing risk in pregnancy and childbearing with increasing age.

9. In only 47 per cent of the maternal deaths was the outcome of pregnancy a live birth, while in 21 per cent there was a stillbirth and in 32 per cent death occurred either before delivery or before the age of viability.

10. The risk of pregnancy and childbearing as computed was lower in the second to fifth pregnancies than in the first or later ones. Pregnancies of higher order than the fifth appear to involve a risk greater than the first.

11. The estimated specific puerperal mortality among "urban" women was 5.7 per 1,000 live births and 4.7 among women in rural areas. Practically all the difference was found in excess in mortality from puerperal sepsis and abortion in the urban group.

12. By type of delivery, the estimated specific mortality was 8.2 per 1,000 live births in the "operative" group and 2.3 in the spontaneous group.

13. The specific puerperal mortality rates for hospital (only those delivered in hospital and dying there) and domiciliary cases were 5.3 and 2.3 per 1,000 live births respectively. On the same basis, the death rates from puerperal sepsis were 1.7 and 0.7 per 1,000 live births respectively.

14. In 18 per cent of delivered cases a Caesarean section was performed. The incidence of Caesarean section among fatal delivered cases was found to be more than twice as high among urban than among rural mothers.

15. Forty-one per cent of the cases for which questionnaires were returned (excluding abortions and ectopic pregnancies) had what was considered adequate prenatal care. Thirty-three per cent received no prenatal care. Forty-four per cent of the rural cases and 24 per cent of the urban group had no prenatal care.

16. In 17 per cent of the 257 cases (excluding those in which no data were given) in which a questionnaire was available, the health of the mother before pregnancy is described as "poor" or "very poor".

17. In only 8 out of 37 deaths from puerperal haemorrhage for which data were available was it stated that a transfusion was given at any time during the conduct of the case. Retained placenta was mentioned in five cases.

18. Of the 61 deaths from puerperal septicaemia studied in detail, there were 5 cases of retained placenta. Information on date of onset varied widely, the 2nd day post partum being the most frequent.

19. In 8 of the 59 cases of puerperal eclampsia reviewed, a physician was called only when the patient was in convulsions. Of the cases delivered, almost a third were delivered by Caesarean section.

20. In 4 of the cases studied, the patient was attended at labour by a midwife.

What the Dairyman Expects from the Medical Officer of Health*

W. H. FORSTER

President of the National Dairy Council of Canada

IN presenting this paper I wish to express the thoughts and desires of the better class of dairymen, both producers and distributors. Just as there are many degrees of efficiency in human endeavour and varying grades in the quality of merchandise, so there are many grades of efficiency among dairymen and many grades in the sanitary quality of milk offered for sale to-day. Fundamental in our consideration of this subject is the provision of an adequate system of supervising the milk supply. It is therefore profitable for us to review briefly the major requirements in a practical and efficient plan. It is the plan followed by the efficient medical health officer in supervising the milk sold in his municipality.

The supervision of a milk supply is a two-stage process covering, firstly, the production at the farm and, secondly, the processing of the milk by the distributor.

Supervision of the Production

The efficient medical health officer sees that the farmer exercises intelligent care and scrupulous cleanliness. He or his inspector visits the farm frequently, for only by close and frequent supervision can he be certain that the cattle are healthy and clean and properly fed. Milk utensils, such as pails, coolers, etc., must be of good construction, with all seams flushed with solder, and must be properly cleaned. Milking must be done by a clean person in a cleanly manner. Milk must be properly strained and cooled to 50°F. in a clean milk-house. During transportation to city or town it should be properly covered to protect it from either heat or frost. Milk inspectors must be trained to be not only law enforcers but educators as well. The farmer does not always realize the importance of some of the regulations for producing high-grade milk and the inspector must know how to teach him to get the best results.

Supplementing the observations of the inspector are the findings of various tests performed in the laboratory or in the field which give information as to the physical and sanitary quality of the milk before it is processed in the dairy. The sediment test affords a striking demonstration to the farmer of the presence of dirt in the milk. The use of a thermometer is invaluable in accurately observing the temperature of the milk. Field tests may be made to determine if the cattle are afflicted with mastitis or other udder infections. By the use of the methylene blue test the inspector may learn if the milk has been produced in a sanitary way and properly cooled. He may also conduct readily a test to estimate the acidity of the milk, giving again an indication of the degree of care exercised in its production. The Babcock test permits of the determina-

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tion of the butterfat content, and the lactometer, in conjunction with this test, will show whether or not the milk has been adulterated.

The trained inspector should be able to make the above observations. In the laboratory, bacteriological examination of milk may be conducted, but the necessary facilities are not usually available except in large centres of population.

SUPERVISING THE DISTRIBUTOR AND HIS PROCESSING METHODS

Having determined that high quality milk is being delivered to the distributor, the first essential is to examine and inspect thoroughly the building and plant of the milk distributor, to make sure that the building and equipment are of proper construction. Floors should be properly graded to trapped sewers, having at least one inch of fall for each five feet of distance from the sewer. Floors should be of cement or tile or some other non-absorbent material. Wood is insanitary as it absorbes milk and becomes foul. The walls and ceilings should be of cement plaster and well painted. Washing operations of bottles and cans should be carried on in a separate room, away from the pasteurizing, cooling and bottling rooms. All apparatus should be so constructed that there are no crevices or seams difficult to clean. Nothing but modern, sanitary pipe should be used to convey milk from tank to tank or from pump to various vats or coolers. The pasteurizing and bottling machines should be of such capacity as to handle the milk in a reasonable length of time. The pasteurizing temperature should be at least 142°F., held for 30 minutes. After pasteurization, sufficient refrigeration must be supplied, either by ice or a refrigerating plant, to cool the milk adequately to 40°F. It should then be held in a refrigerator at 40°F. or lower. Pasteurizers should be equipped with recording thermometers so that temperatures can be checked by the inspector. Scrupulous cleanliness on the part of the operator and all other employees must be exercised in the handling of the products within the dairy at all times. The equipment should be thoroughly washed and sterilized at the conclusion of the day's operations and re-sterilized in the morning before operations are begun. The inspector in charge of this supervision must be a properly qualified man with sufficient technical knowledge that he can teach any operator who is not following the best dairy practice how to improve his methods.

From these observations you will note that it is necessary for the medical health officer to have considerable knowledge of the dairy business. If an inspector is employed, he should be fully qualified and able to supervise intelligently the production of milk on the farm and the handling of it in the plant. To do this, he must be well trained. The conscientious dairyman is as much interested in the protection of the public health as is the health officer and he looks to the health officer to see that all those engaged in the industry conform to the best practice. This can be done only when those responsible have the proper qualifications and facilities and give sufficient time to this work.

OBSERVATIONS ON MILK SUPERVISION IN ONTARIO

In order to give a picture of the efficiency or inefficiency, whichever the

reports show, of some health departments in Ontario in supervising the production of milk, extracts from letters received after enquiry among dairymen in a number of cities and towns are presented.

The following comment on the work of the health officer and his department is a sample of the observation made by dairymen in four or five of the best supervised cities in Ontario.

"We have a very efficient health department in this city and cannot think of any specific practical suggestion of improvement to offer at this time."

Conditions in these cities, however, are not as satisfactory as they could be. Frequently even in large cities with well organized health departments milk inspectors are appointed who have very little knowledge of their work and apparently receive little or no training prior to taking up their duties. Such an untrained inspector may leave a very bad impression and reduce the dairy's respect for and confidence in the local board of health.

The following extracts are from letters received from various localities in Ontario in which the dairymen are not satisfied with the supervision provided by the medical officer of health and his staff. The quotations must not be considered as reflecting on the health officer, but rather on our present provision of health supervision by the system of part-time health officers. The medical health officer is a physician usually engaged in private practice and trained in the treatment and prevention of disease. He has had, usually, very little opportunity to learn the modern methods used in the proper safeguarding of milk. Further, in the majority of municipalities he receives such a pittance in the way of remuneration for his services as health officer that he feels his duty reasonably well done when he deals with communicable diseases as reported to him, exercising all due precautions for the prevention of their spread. Having carried out his more strictly medical duties, he expects that the milk inspector usually appointed by the local municipality should take care of the milk business of the town.

Quotation from Central Ontario "The sanitary inspector, while he is a good fellow to meet in a social way, has no qualifications and has had no training for the position. Our medical health officer is a most affable man. I believe his salary is something like $500.00 per year, and that pretty well explains the half-heartedness that he displays in such a contentious matter as the pasteurization of milk. Personally he is for it. We had an epidemic of typhoid fever which was traced to a raw milk source. A hired man on the farm happened to be a typhoid carrier."

In this town the inspector each month asks the milk distributors to leave a sample of the milk on his doorstep, as it is inconvenient for him to go to their plants. Yet this city has a by-law with the strictest regulations covering the sanitary care of the cows and stables, food and water, with minute instructions on the care of the milk, the utensils, floors, milk vats, cans and bottles, and all handlers of milk.

Quotation from Northern Ontario "I do think that the medical officer of health should be a full-time officer, or one who is in no way engaged in private practice, in which case he is likely to be prejudiced in favour of those dairymen who are his patients. The health inspector should be qualified by proper training. More regular inspection should be made of the good as well as the not-so-good dairies. We are flattered to

think that we have not had an inspection for over a year, but we should have had one. Milk samples should be taken by the inspector, and not handed to him."

From an Eastern Ontario Town "To my knowledge, in three years here inspection of dairies has been made once. There are no inspectors apart from the medical officer of health. There is no inspection of milk from any of the distributors whatever, for sediment or bacteria. Plant and dairy inspection is nil."

From a Western Ontario City "The position in reference to our city is that the milk inspector is a part-time man and needs other practice to make a living. He is a young man and would do good service, provided he had the backing, but in the past the city councillors have not always taken the advice of the Board of Health."

Here is a health officer willing and trying to improve conditions, but prevented by his municipal council.

Report from an Eastern Ontario Town "I have been in business twelve years and at no time have I had an inspection, either by the medical health officer or the sanitary inspector, other than two or three butterfat tests a year. This operation is completed at one of my competitors' plants, owing to the inspector's inability to do the same."

This writer cites insanitary methods of some dealers and concludes by saying: "With so many numerous insanitary methods of handling milk, it is high time this condition is brought before the proper authorities."

Another observation made by a most reputable dealer in eastern Ontario was this:

"My own criticism of some medical health officers is that they compel some distributors to observe every detail of the municipal by-law and permit others to sell milk the quality of which is not up to the standard required by the law."

From one central Ontario city I received a very gloomy letter from a man who I personally know is trying to put out a high-grade article. He complains bitterly of the lack of effort on the part of the health department to remedy a situation which, according to him, is sadly in need of it. I would hesitate to quote some of his statements describing the lack of sanitation in the industry in his town. The writer reviews twenty years of lack of effort of the medical health department to improve the milk in his town and concludes thus:

"The sanitary inspector is still on the job, and anyone applying for a licence to sell milk gets it by paying $2.00. As far as I know, his farm is never looked at again. During the past two years the city has not thought it worth while to set apart any money to have the sanitary inspector go out and inspect the farms again."

A leading dairy paper distributed in Ontario, *The Ontario Milk Producer*, in discussing the milk situation in one Ontario town, concludes with this statement: "There is no inspection either of producers' or distributors' premises."

A most reputable dealer, well known for his sane and careful judgment, makes this observation:

"Inspection by medical health officers in small towns and villages is a farce, because of the lack of training on the part of the officer. My thoughts are: license all dairies; employ as inspectors practical plant men, men who know. The medical officer of health in this town, a local doctor, is doing the best he can; handicapped, however, because of his lack of knowledge of dairy work."

I could quote many more statements by dealers all over Ontario which show gross inefficiency of milk inspectors and seeming neglect of the sanitary supervision of the local milk supply, but I have quoted sufficient to show you that the better class of milk distributors, from whom these observations have been gathered, are not satisfied that the milk industry in their towns is receiving the attention that it should receive from the medical officers of health and their inspectors.

The importance of the employment of trained inspectors is evident, for it is appreciated by the better producers and distributors in the dairy industry that there are many producers selling milk in small towns and cities who are not properly qualified or equipped to produce sanitary milk, and that there are milk distributors operating pasteurizing plants who have inefficient equipment, who have not sufficient dairy training to operate a plant properly, and who need supervision and education. The town policeman, the janitor of the town hall, and the garbage collector have been known to be appointed milk inspectors. Such men—and it is not their fault—with no previous experience in sanitary health work, cannot adequately supervise and deal with the sanitary problems of milk supply. Thus there is a great need for qualified milk inspectors.

THE NEED FOR GOVERNMENT SUPERVISION

What about the pasteurization of milk? Are you in favour of it? Let us examine the experience of municipalities that have pasteurization and of those that have not. In an address before the Rotary Club in Toronto, Dr. Gordon Bates, General Director of the Canadian Social Hygiene Council, stated:

"Toronto has pasteurized its milk since 1914. During the last twelve years there has not been a single case of bone tuberculosis of bovine origin originating in the city of Toronto. Such cases as have come in have originated outside Toronto. Similarly, gland tuberculosis of bovine origin has all of it come from outside Toronto. It is well known that pasteurization of milk will definitely prevent this type of disease. Incidentally, pasteurization of milk will also prevent the spread of typhoid and paratyphoid fever, scarlet fever, septic sore throat, and diphtheria. You will remember that the most notorious typhoid epidemic in the history of this country, that of Montreal, with 5,000 cases and 500 deaths, resulted from failure to institute proper pasteurization."

Dr. James Roberts, Medical Officer of Health for Hamilton, said:

"I am glad to inform you that our records over the past ten years do not give evidence of deaths of children from bone tuberculosis. There are a few cases of adults dying from Potts disease of the spine, which, however, probably had its origin years before. . . . . During the same period there has not been a case or death from typhoid fever attributable to milk. . . . . As early as 1924 99 per cent of all milk in Hamilton was pasteurized and from 1928 pasteurization has been compulsory."

Dozens of the most eminent authorities in Canada and the United States recommend and urge pasteurization of milk by qualified operators and checked by efficient inspectors.

In municipalities where pasteurization is not compulsory, it will be of interest to note a few of the epidemics that have occurred in Ontario in the last few years which have been traced mostly to raw milk.

In 1921, at Vineland, there were 25 cases of typhoid fever due to a typhoid carrier delivering milk. There was no supervision of the milk supply.

In 1923, at Arnprior, 7 cases of typhoid fever. A typhoid carrier was handling raw milk. Again there was no supervision of the supply.

In 1925, at Vineland, 11 cases of diphtheria attributed to a carrier handling raw milk.

In 1927, in a large town in western Ontario, 109 cases of typhoid fever and 7 deaths. These occurred on one milk route. Pasteurization was supposed to be in effect but was incomplete, owing to lack of knowledge on the part of the operator.

In 1930, in an eastern Ontario town, 18 cases of typhoid fever and 3 deaths, caused by the raw milk supply from one dairy. A milker on one of the producers' farms was responsible.

In 1930, in northern Ontario, 457 cases of septic sore throat traced to one raw milk supply.

In 1931, in a fairly large city in southern Ontario, an outbreak of 457 cases of paratyphoid fever on one milk route. The milk was supposed to have been pasteurized; again, insufficient knowledge on the part of the operator.

There are many other cases that might be cited but these are sufficient to show the necessity for, firstly, in the raw milk instances, pasteurization as a safeguard and, secondly, qualified, certificated plant operators and the selection of properly trained dairy inspectors instead of inexperienced persons appointed by the city or town council and put in charge of our most perfect and valuable food.

If compulsory pasteurization is not in effect in your municipality, the raw milk supply should be a Grade "A" milk, the specifications for which are, briefly: cows must pass the tuberculin test and the blood test for abortion; stables and equipment must meet an established standard; and the sanitary care of the milk must be such that the bacterial count will not exceed 50,000 per cc. of milk.

The Provincial Government requires all butter and cheese factories to be in charge of a properly qualified operator who must hold a certificate of qualification from the Dairy Department and the department must have qualified inspectors to check the qualified operators, yet milk plants can be opened with no requirement of qualification from owner or operator. Milk is one of our most valuable and delicate of foods, requiring more care and supervision than butter or cheese; yet, as stated, anyone can engage in the milk business without previous knowledge or experience and get a licence from some boards of health who have not even a properly qualified inspector to check the operations of this inexperienced dairyman. Is it any wonder that we have epidemics of disease caused by dirty milk? When are we going to wake up and demand that our milk producers receive proper inspection at the farm and that our milk distributors have properly qualified plant operators who can show certificates of efficiency from the Provincial Department of Agriculture or Health, as may be arranged? Your waterworks plants must first get the approval of the sanitary engineers of the Provincial Department of Health. Why should not milk plants be approved by qualified dairy engineers before they are permitted to operate? It is equally important to have trained inspectors to supervise milk production and milk-plant operation as it is to have waterworks engineers, plumbing inspectors and building inspectors, and such men are all selected because of their special qualifications and knowledge of the work that they supervise. Are *your* milk inspectors so selected? The consumers in your town depend on you, as the medical officer of health, to protect and safe-

guard their milk supply and if something happens in the way of an epidemic, some morning you will be asked, "What is the matter with our health department?" Many of these past epidemics might have been avoided if the local municipal council had listened to the advice of their medical health officer and passed a pasteurization by-law which would have protected the citizens against such outbreaks. We will not have an efficiently supervised milk supply in the province of Ontario until the control of the milk business is taken out of the hands of municipal politics. Our present system is wrong.

CONCLUSIONS

All these observations have led me to draw the following conclusions. Legislation should be passed to bring about these much needed reforms:

1. *Compulsory pasteurization.* We should have compulsory pasteurization in the province of Ontario.

2. *Competent inspectors.* Our staff of milk inspectors should be men trained for their work in the same way that you, as a medical health officer, have been technically trained in the prevention and treatment of disease, and hold certificates of efficiency. These milk inspectors should have a special course, to be taken at the Ontario Agricultural College, which would especially fit them as inspectors for the supervision of the production of milk on the farm and for the supervision of processing plants in towns and cities.

3. *Licensing of plants and operators.* Every plant operator should be licensed to operate a plant only when he has received a certificate of qualification from the government, a requirement now made of cheese and butter makers.

4. *Full-time health services.* Where towns and cities are not large enough to employ a full-time inspector the province should be divided into districts and a full-time qualified medical health officer put in charge of each district, the size of which would be governed by the number of urban municipalities requiring supervision.

5. *Provincial control.* The supervision and control of the milk business should be taken out of the control of municipal politics and put under the control of the Ontario Department of Health. The reason for this recommendation is that many medical health officers and boards of health have recommended pasteurization and other advanced methods of sanitary control of the milk industry and have had their recommendations turned down by the municipal council, thus handicapping the local medical health officer in the protection of the public health in his community, and at the same time disheartening him in the work he is so faithfully trying to perform in the interests of the public health.

The National Dairy Council of Canada, the Ontario Milk Producers' Association, and the Ontario Milk Distributors' Association are aiming at higher standards in production, processing and supervision of our milk supply. For these organizations I pledge to you, as health officers, their heartiest support of anything that you can do which will raise the standard of the supply of milk for the citizens of this province to a higher level.

The Prevention of Scarlet Fever in a Children's Hospital*

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SINCE Sydenham's description of scarlet fever, as it prevailed in London during the years 1661 to 1675, our knowledge of the clinical picture of scarlet fever has remained practically unchanged. The development of further knowledge of scarlet fever began some 200 years later when the role of bacteria as causative agents in communicable diseases began to be appreciated. To-day it is generally accepted that the etiological agent for scarlet fever is a streptococcus. This being so, scarlet fever, erysipelas and puerperal sepsis are in the same category and may be regarded simply as different manifestations of a streptococcal infection developing under special conditions of tissue susceptibility. Scarlet fever therefore may be looked upon as a particular manifestation of a streptococcal infection characterized by a group of clinical signs and symptoms. Bacteriologically the clinical course and complications of this disease are produced by one or more of three factors: (a) the invasion of body tissues by the toxins of the scarlatinal streptococcus, (b) the invasion of body tissues directly by the scarlatinal streptococcus, and (c) the invasion of body tissues by a secondary infection.

In 1923 George and Gladys Dick published the results of a series of experiments showing that a specific type of haemolytic streptococcus was the causative organism of scarlet fever. In their original work they isolated and used two strains now known as Dick I and II. They further demonstrated that this streptococcus produced a potent soluble toxin which was responsible for the toxaemia, nausea and rash of scarlet fever, and that recovery from this disease with subsequent immunity depended on the production of antibodies, particularly antitoxin. Since 1923 it has been shown that there are innumerable strains of haemolytic streptococci, scarlatinal and non-scarlatinal, the majority of which produce a toxin. The toxins of these individual strains of haemolytic streptococci have been shown by several workers to differ quantitatively and qualitatively. These differences are by no means clear-cut and do not constitute a means of distinguishing scarlatinal strains. This indefinite relationship of haemolytic streptococci and other toxins to one another may help to explain some of the difficulties encountered in the Dick test, in active immunization, and in the use of scarlatinal streptococcal antitoxin.

The reliability of the Dick test for determining the susceptibility of individuals to scarlet fever has been shown by the results reported from many sources. Its value depends on the material used and the technique employed,

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which must be exact in every detail. Performing and interpreting this skin test should be done only by those trained in this procedure. A negative Dick test has been shown to have a decided value and is a very important factor in immunization against scarlet fever.

Immunity to scarlet fever has been extensively investigated. New-born infants frequently show negative skin reactions which become positive during the first year of life. The incidence of natural immunity to scarlet fever after early infancy depends on conditions which favour exposure to scarlatinal infection. Age and sex are not directly related to this immunity.

Acquired immunity presents itself in two forms, active and passive. Passive immunity is made possible through the introduction of scarlet fever antitoxin. It has been shown that 5 cc. (2,500 units) of this antitoxin inoculated into a positive skin-test reactor will produce a passive immunity within a few hours, lasting from 7 to 10 days. Serum reactions, both local and general, following its use may occur in from 30 to 40 per cent of the cases. When deciding upon the value and use of antitoxin in passive immunization it is well to consider a few important points: (1) the short period of protection given by this serum; (2) the frequency of serum reactions, at times severe; (3) the many individuals who have acquired an active immunity to the scarlatinal streptococcus at some previous period, unknown to themselves; (4) the prevalence of the various streptococcal infections; and (5) the possibility of rendering these individuals sensitive to horse-serum. These factors related to passive immunization with scarlet fever antitoxin should be familiar to the medical profession, especially those in general practice, before they adopt this form of prevention in scarlet fever.

Of more importance is the value of active immunization. Some degree of reaction, local or general, is to be expected in many adults, following one or more doses of the toxin. Fewer reactions occur in children. The reactions vary from local redness or oedema to general symptoms of malaise, headache, vomiting, fever and a scarlatinal rash.

Institutions, especially where children are being cared for, provide a suitable field for the study of scarlet fever in its various aspects. The scarlatinal streptococcus is admitted frequently in some form to those institutions. Many carriers of this streptococcus, and mild or missed cases of scarlet fever, are unknowingly admitted to the wards of a children's hospital. The infection spreads rapidly and soon causes disorder in the routine of the institution. It gives rise to a serious situation, both administratively and financially.

Isolation of patients, quarantine of contacts and other precautions have been followed for many years and have not met with any marked degree of success. With the introduction of immunization as a measure for the possible prevention and control of scarlet fever, it was soon adopted as a routine measure in many institutions. In September, 1924, immunization for scarlet fever was begun in the Hospital for Sick Children, Toronto. Cases of scarlet fever were admitted to hospital only when justified by the seriousness of existing complications. These latter cases and those developing scarlet fever after admission

to hospital were isolated in an infectious ward, a separate unit of the hospital. The quarantine of contacts and other precautions were discontinued throughout the hospital, except in the infectious ward. The entire personnel of the training-school for nurses, the house-staff, patients in hospital and employees of the hospital were included in this programme of immunization.

Immunization of Nurses

The nursing-staff of the Hospital for Sick Children consists of two groups, nurses taking the regular course of three years' training and nurses sent from affiliated hospitals for a period of three months' training in the Hospital for Sick Children. In 1925 the affiliated hospitals were requested to perform Dick tests and actively immunize susceptible nurses before they were transferred to the Hospital for Sick Children. On investigation it was found that from time to time this work was incompletely carried out. In the following table the regular staff and affiliates are therefore tabulated as separate groups. A Dick test was performed upon each nurse when she entered the hospital in her probation period. Positive skin-reactors were given a course of active immunization, consisting of 5 doses (4 doses until July, 1927) of scarlet fever toxin containing 330, 1,000, 2,300, 5,000 and 10,000 skin-test doses, at weekly intervals, and followed by a second Dick test two weeks after the last dose. Additional doses were given to those remaining Dick positive until a negative Dick reaction was obtained. These preventive measures were also adopted in the case of the house-staff and all employees of the hospital.

The results of the immunization of nurses, begun in 1925, are presented in Table I.

TABLE I

INCIDENCE OF SCARLET FEVER AMONG NURSES
HOSPITAL FOR SICK CHILDREN, TORONTO
1918-1933

	H.S.C. Nurses	H.S.C. Nurses with scarlet fever	Case Rate per 100 nurses	Affiliated Nurses in school	Affiliated Nurses with scarlet fever	Case Rate per 100 nurses
1918-24	687	42	6	648	25	3.9
1925-33	1,054	1*	0.09	1,920	15	0.78

*This nurse was Dick negative, was not retested and contracted scarlet fever the following year. Two probationers developed scarlet fever within a few days of admission before they had had Dick tests or immunization; two other nurses developed scarlet fever during the course of active immunization before they had received the full number of doses. These 4 cases are omitted from the table.

Immunization of Patients

As a routine, the Dick test was performed on all patients within 24 hours of admission and those reacting positively were passively immunized with antitoxin. Until 1930 the quantity of antitoxin used was 2 cc., but since it appeared that in 10 patients this amount was insufficient, the dose was increased in 1930 to 5 cc., which represents not less than 2,500 units. In patients remaining in the hospital more than 10 days, either the dose of antitoxin was repeated or active

immunization with toxin was begun. Since 1930 one nurse has been responsible for the Dick testing of all patients on admission. It is the duty of the head nurse of each ward to see that passive or active immunization is given by the interne to all patients with Dick positive reactions. Diabetic, nephritic, and asthmatic patients are not given antitoxin but are isolated in a separate room if Dick positive. Infants of eighteen months or younger are treated in a separate unit of the hospital and are not included in the preventive scheme.

The nurse responsible for the Dick testing of patients is also responsible for the Dick testing and active immunization of all dietitians, nursery-maids, maids, orderlies and any others who might come in contact with the patients in the wards. The nurses are not included in this group.

A study of the cases of scarlet fever treated in the hospital during the years since preventive measures were instituted (namely, from 1925 to 1933) shows the value of these procedures. During this period the total admissions were 57,266. Of the 169 cases of this disease treated during this period, 127 were known to have scarlet fever at the time of admission or were diagnosed as scarlet fever cases within 3 or 4 days of admission. These cases may be considered in this discussion as constituting group 1. Those who developed the disease later than 3 or 4 days after admission numbered 42; this group includes patients admitted with the diagnosis of "burn" at the time of admission and who subsequently developed scarlet fever. Since group 1 consists of patients who either were admitted with a diagnosis of scarlet fever or were presumably in the invasive stage, the preventive measures were applicable only to the 42 patients in group 2. This group may be divided as follows:

(a)	Patients who received no Dick test and no antitoxin	11
(b)*	Patients with a positive Dick reaction who received no antitoxin	10
(c)	Patients who received a small dose of antitoxin (2 cc.)	10
(d)	Patients who received antitoxin but in whom the dose was not repeated in 10 days and who were not actively immunized	2
(e)	Patients who developed scarlet fever during the course of active immunization	2
(f)	Patients with a negative Dick reaction and who were not immunized	4
(g)	Patients who developed scarlet fever although a full dose of antitoxin (5 cc.) was given	3
	Total	42

*Group (b) includes one diabetic and two asthmatic patients who consequently received no antitoxin but were isolated. The remainder of group (b) and group (a) received no antitoxin through carelessness.

From a study of the above analysis of the 42 cases occurring in the hospital in spite of a programme of active and passive immunization it is evident that in all but 7 cases a reasonable explanation of the occurrence of the disease, in so far as immunization procedures are concerned, is given. Of the 7 cases only 3 had received a full prophylactic dose of antitoxin; the other 4 were not immunized because of the recording of a negative Dick test.

Scarlet Fever Complicating Burns

Owing to the frequent occurrence of scarlet fever as a complication of

burns, all patients admitted with a diagnosis of "burn" are given 5 cc. of antitoxin within 24 hours of entering the hospital. Until 1930 the quantity of antitoxin given was 2 cc., at which time it was increased to 5 cc. since the smaller amount appeared to be insufficient in some cases. The following table summarizes the incidence of scarlet fever in cases of burns before and after the institution of preventive measures.

TABLE II

INCIDENCE OF SCARLET FEVER AMONG CASES OF BURNS
HOSPITAL FOR SICK CHILDREN, TORONTO

	1917-1924	1925-1933
Number of cases with burns admitted to hospital	406	680
Number of cases with burns developing scarlet fever	25	7
	(6.15%)	(1.03%)

Excluding the cases which did not receive proper immunization, no cases of burns developed scarlet fever from 1925 to 1933.

Of the 7 cases developing scarlet fever, 5 did not receive antitoxin. These cases occurred from 1925 to 1930. The remaining two, occurring in 1927 and 1929, received only 2 cc. of antitoxin. No cases of scarlet fever complicating burns have occurred in the past four years. It is interesting to note that every patient who failed to receive antitoxin developed scarlet fever.

SUMMARY

The incidence of scarlet fever among hospital patients, nurses and other hospital personnel may be reduced greatly by the use of the present methods of specific immunization against this disease. This is shown by the experience of nine years in the Hospital for Sick Children.

Passive immunity is conferred within a few hours by the administration of 2,500 units of scarlet fever antitoxin. This immunity lasts from 7 to 10 days.

The administration of 5 doses of scarlet fever toxin will actively immunize a high percentage of susceptible individuals.

Local and general reactions, at times severe, occur in many cases following both passive and active immunization.

The value of the Dick test in a programme of immunization is indicated.

This work should be conducted only by those specially trained in its technique. Periodic retesting of persons actively immunized should be carried out, and careful records of results obtained, if the ultimate value of these methods is to be estimated.

Recent Advances in the Treatment of Cyanide Poisoning*

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ALTHOUGH mortality statistics show that cyanide poisoning is not of great importance as a cause of death, the spectacular nature of many of the deaths from cyanide ingestion or inhalation keeps this type of poisoning constantly in the minds of the public. The deaths from cyanide poisoning which do occur are suicidal or are due to accidents in fumigating or in connection with the various industrial and laboratory uses of cyanide. There are also occasional cases of poisoning from plant sources.

Interest in antidotes for cyanide was stimulated by Sahlin's[1] discovery, in 1926, of an antagonism between methylene blue and cyanide. Recent work has shown that there are other, more potent antidotes than methylene blue. The experimental results which have led to an understanding of the mode of action of methylene blue and to the discovery of these other antidotes, as well as the clinical use of methylene blue, are described in this paper.

In discussions of acute poisoning, carbon monoxide and cyanide are often associated because both act, essentially, by producing asphyxia. This association has led to the trial of nearly all the new cyanide antidotes in the treatment of carbon monoxide poisoning. Although these experiments have met with little success, they will also be considered.

Cyanide Poisoning

Cyanide poisoning in man is a very acute condition. According to Sydney Smith, hydrocyanic acid causes death in from 10 seconds to 2 hours, with an average time of 2 to 10 minutes. As a rule, potassium cyanide is slower in producing symptoms and causing death. The symptoms are loss of consciousness, muscular twitchings and convulsions. The eyes are fixed and glassy, the skin moist and clammy, pulse almost imperceptible and, in the later stages, respiration is spasmodic. Death follows due to cardiac and respiratory failure. That the condition is due to the inability of the tissues to use oxygen, rather than to a deficient oxygen supply, is shown by the fact that the venous blood is bright red and fully oxygenated when death occurs. There is probably also a direct action on respiratory movements by way of the carotid sinus, but this is of secondary importance. There is no evidence that the toxic action of cyanide *in vivo* is due to an action on the blood, as is the case with carbon monoxide.

The action of cyanide on respiration *in vitro* has been repeatedly studied. There is fairly general agreement that cyanide inhibits tissue respiration. It probably does this by combining with some iron compound in the cell which is active in catalysing cell oxidations. It is suggested that this com-

*Presented at a meeting of the Toronto Physiological Society, February 12, 1934.

pound is the same iron porphyrin with which carbon monoxide combines, but this is not definitely proven. Some workers suggest that cyanide must combine with other iron compounds in addition to the respiration enzyme and probably also with various organic sulphur compounds.

Since it is clear that cyanide exerts its toxic influence by interfering with the mechanism of cellular oxidations, it follows that any antidote for cyanide poisoning must have one of two actions. Either the antidote must remove or detoxify the cyanide, or it must replace the inactivated catalysts. The fact that methylene blue can act as a catalyst for certain biological oxidations has led to its use as an antidote for cyanide poisoning. It seems certain that the workers who first used methylene blue for this purpose felt that it came in the second class but present evidence suggests that all the known antidotes for cyanide act by removing or detoxifying the cyanide.

Methylene Blue

In general, it may be said that methylene blue stimulates respiration in isolated cells and tissues, although there are several exceptions to this statement. It has also been shown that the injection of methylene blue leads to an increase in metabolism and a rise in body temperature in the intact animal.

There is much evidence to show that methylene blue will reverse the inhibiting effect of cyanide on cellular oxidations. There is considerable disagreement concerning the extent of this antagonism. This effect of methylene blue is complicated by its stimulating action on normal tissue oxidations.

The evidence that methylene blue antagonizes the action of cyanide in the intact animal is overwhelming. Apparently the first experimental demonstration of this action was made by Sahlin in 1926, using rats. Since that time the observation has been confirmed on dogs, rabbits, rats and mice, by a considerable number of workers. Trautman alone questions the efficacy of methylene blue. He found that it was of no value in the treatment of hydrocyanic acid gas poisoning when the animals used had inhaled a lethal or sub-lethal dose of the gas. Much of this work served merely as a qualitative demonstration of the antagonism but some of the more recent work has been quantitative. Hug[2] determined the minimum lethal dose of neutralized sodium cyanide when given to a dog subcutaneously. He then found that methylene blue would protect dogs against 1½ to 2 minimum lethal doses of cyanide but not against 3 minimum lethal doses. This observation has been confirmed on several occasions; there are no reports of protection against much larger doses.

In view of this unquestionable evidence of an antagonism between methylene blue and cyanide both *in vivo* and *in vitro*, there has been much speculation concerning the mechanism of the antagonism. There are two possibilities. The methylene blue restores the oxygen uptake either (1) by replacing the inactivated respiration enzymes, or otherwise stimulating cellular oxidations, or (2) by removing or detoxifying the cyanide.

The evidence that, in the intact animal at least, methylene blue acts

by actually removing the cyanide from the tissues is now fairly convincing. Since methylene blue and cyanide do not combine directly, it is necessary to bring in some other substance. This substance is methaemoglobin. The present view is that, in the intact animal, methylene blue acts on the haemoglobin of the blood to form methaemoglobin which then combines with cyanide from the blood and tissues to form the stable and relatively non-toxic compound cyanmethaemoglobin, thus freeing the tissue respiration enzymes and allowing oxidations to proceed. This view is based on the following facts which have been established by recent investigations:

(1) Methylene blue does act on haemoglobin to form methaemoglobin both *in vivo* and *in vitro*.

(2) Methaemoglobin and cyanide combine very rapidly even at room temperature to form a stable compound known as cyanmethaemoglobin.

(3) Other methaemoglobin-forming substances and methaemoglobin itself are good antidotes for cyanide poisoning. Among the methaemoglobin-forming substances that have been found to be active are sodium nitrite, amyl nitrite, pyrogallol, pyrocatechol and phenylhydrazine.[3] Of these, both amyl nitrite and sodium nitrite have been shown to be more effective than methylene blue (see Table I).

(4) There is direct evidence that methaemoglobin can actually reverse cyanide inhibition of respiration in isolated tissues.[4]

It is obvious that the efficacy of any methaemoglobin-forming antidote is strictly limited by the amount of haemoglobin that can be safely converted into methaemoglobin. Experiments on aniline dye poisoning show that conversion of two-thirds of the haemoglobin to methaemoglobin is fatal, in dogs. Wendel[5] has calculated that this proportion of methaemoglobin in the blood of an average man would only be sufficient to combine with about four minimum lethal doses of cyanide. This calculation is in agreement with most of the experimental results although some workers have obtained protection against larger doses by the use of nitrites.

The experimental evidence available indicates definitely that nitrites are likely to prove more satisfactory for clinical use than methylene blue. Due to the ease and rapidity with which it may be administered by inhalation, amyl nitrite seems to be the most suitable. Chen, Rose, and Clowes[6] have outlined the actual method which might be followed in treating a case with amyl nitrite.

The Detoxification of Cyanide

The mechanism which has been outlined for the alleviation of cyanide poisoning by the removal of the cyanide from the tissues to the blood, has the disadvantage that it leaves the victim full of cyanmethaemoglobin. Hug has shown that this is merely the conversion of an acute into a slower and more prolonged intoxication. However, this temporary respite gives the detoxifying mechanisms of the body time to act. The ability of the body to detoxify small amounts of cyanide has been noted on many occasions. If the cyanide is administered sufficiently slowly, many times the minimum lethal dose can

be detoxified without producing any ill-effects. Hug[7] has shown that rabbits are able to detoxify cyanide at the rate of about 1 mg. per kg. of body weight per hour.

There is little doubt that this detoxification is brought about by the conversion of the cyanide to thiocyanate. As a result of their own and other experiments Smith and Malcolm[8] conclude that: "If thiocyanate formation is not the only method of cyanide detoxication, other processes are very secondary in importance to it."

There is evidence that the reduction of methaemoglobin can take place in the blood. It is probable that this reduction is accomplished by the simultaneous oxidation of some carbohydrate. The spleen may play an active part in this process.

It appears therefore that not only can the body detoxify and excrete the cyanide in the cyanmethaemoglobin but it also can restore the lowered oxygen capacity of the blood, thus bringing about complete recovery.

Sulphur-Containing Compounds

This discussion of the detoxification of cyanide by conversion to the thiocyanate leads naturally to a discussion of that group of cyanide antidotes which contain sulphur. As far back as 1894 Lang showed that various sulphur compounds had an antidotal action. In 1897, Heymans and Masoin showed that sodium thiosulphate was a preventive of cyanide poisoning. Recent work has revived this interest in sulphur compounds. Hug found that sodium thiosulphate was a very effective prophylactic for cyanide but not quite as good as methylene blue in treatment. Chen, Rose and Clowes find that both sodium thiosulphate and sodium tetrathionate will protect dogs against 3 minimum lethal doses of cyanide. Hug finds that colloidal sulphur and sodium sulphide are without effect. Cystine, cysteine and glutathione are active *in vitro* but do not seem to have been tried *in vivo*. Turner and Hulpieu,[9] in reviewing the work in this field, state that: "The results taken as a whole show that almost any sulphur compound, which is capable of liberating sulphur in the body and is not too toxic in itself, is definitely protective against cyanides; further that sodium thiosulphate is just as effective if not more so, than other sulphur compounds." It is generally agreed that these sulphur-containing compounds act by forming thiocyanates from the cyanide. However, there is evidence that, *in vitro*, some organic sulphur compounds oxidize cyanides to cyanates. Cyanates are said to be 32 times less toxic than cyanides.

The use of sugars and their derivatives as antidotes for cyanide poisoning deserves mention. There is general agreement that reducing sugars have a detoxifying action *in vitro* but there is some difference of opinion concerning their action *in vivo*. Forst reports protection up to 4 minimum lethal doses with previous administration of glucose or glucose plus insulin. Others have failed to repeat this work. Dihydroxyacetone is said to have a very marked but transient action. By combining it with sodium thiosulphate Forst claims to have protected animals against 10 m.l.d. of cyanide.

From this discussion it is clear that, omitting the sugars, there are two general groups of cyanide antidotes, the methaemoglobin-formers and the sulphur compounds. It is also obvious that the mode of action of the two types is quite different, hence an added effect might be expected when antidotes of the two types are given simultaneously. This idea has been tested experimentally by Hug, Chen, Rose and Clowes, and Buzzo and Carratala. The striking results obtained by Chen, Rose and Clowes[10] are shown in Table I.

TABLE I*

Drug	Number of minimum lethal doses of NaCN detoxified
Nitroglycerine	0
Methylene blue	2
Sodium thiosulphate	3
Amyl nitrite	4
Sodium nitrite	4
Amyl nitrite and sodium thiosulphate	10
Sodium nitrite and sodium thiosulphate	13

These results show that the effect is more than additive; it is in fact an example of synergism or potentiation. Buzzo and Carratala present even more striking results. They claim to have obtained protection up to 10 m.l.d. with sodium nitrite alone and up to 18 m.l.d. with nitrite and thiosulphate together.

Clinical Use of Methylene Blue

Of all the antidotes for cyanide poisoning that have been mentioned, methylene blue seems to be the only one that has been given clinical trial. Geiger[11] in San Francisco has reported the use of methylene blue in three cases of cyanide poisoning with remarkably good results. In all these cases potassium cyanide was taken by mouth. The amounts taken were 15, 16.4 and about 100 grains, respectively. The amount taken could not be accurately verified in the latter case. The first patient was given 50 cc. of 1 per cent methylene blue solution intravenously. The others were each given 100 cc. in two doses. In each case recovery was rapid and dramatic.

In evaluating the evidence already presented for the view that methylene blue acts by the formation of methaemoglobin, it should be noted that Geiger was unable to find methaemoglobin in the blood of the last two of these victims a few minutes after the injection of the methylene blue. However, in one case there was a momentary return of cyanosis after the injection. The expected rise in temperature following the injection was noted in two of the cases.

Sydney Smith states that as little as 5 grains of potassium cyanide has proved fatal to a man. Hence it appears that in the two cases where the amount taken was known with certainty, about three minimum lethal doses of cyanide had been ingested. This dose, in animals at least, falls just at the upper limit of the antidotal capabilities of methylene blue.

*Chen, Rose and Clowes, *Proc. Soc. Exp. Biol. and Med.*, **31:** 252, 1934.

CARBON MONOXIDE POISONING

Carbon monoxide poisoning is a much more important problem than cyanide poisoning. In 1924, Henderson stated that carbon monoxide poisoning was nearly as important, as a cause of death, as diabetes. The effective treatment of carbon monoxide poisoning dates from the year 1814 when Samuel Witters of Dublin first made use of pure oxygen inhalation.[12] He even went so far as to demonstrate the efficacy of this treatment by an experiment upon himself. In spite of the excellent results obtained, it was nearly a hundred years before this method of treatment became general. In 1920, Yandell Henderson recommended the addition of carbon dioxide to the oxygen in order to stimulate respiration. This treatment with carbon dioxide and oxygen has proved very satisfactory and still seems to be the best available treatment for carbon monoxide poisoning. It is, of course, merely a rapid means for ridding the body of the carbon monoxide and involves no detoxification of any kind.[13]

The recent interest in antidotes for cyanide poisoning has led to the application of nearly all the cyanide antidotes to carbon monoxide poisoning. There seems to be no rational basis for this line of endeavour. Carbon monoxide and cyanide both kill by causing asphyxia, but there the similarity in their actions ends. Cyanide acts on the tissues without affecting the blood, whereas carbon monoxide acts on the blood and cannot affect the tissues in the intact animal. It is true that carbon monoxide can act directly on tissue cells to inhibit their oxidations, but a concentration of 80 to 99 per cent carbon monoxide is required for this purpose, while as little as 0.2 per cent of carbon monoxide may produce fatal poisoning in man.

Methylene Blue

Brooks first recommended the use of methylene blue in carbon monoxide poisoning. In experiments on rats she found that the recovery time from a sublethal dose of carbon monoxide was cut in half by the intraperitoneal injection of methylene blue. A similar antidotal action was shown in other experiments on rabbits using lethal doses of the gas[14]. Draize also suggests that methylene blue has a beneficial effect in carbon monoxide poisoning in rabbits.

Haggard and Greenberg[15] object violently to the use of methylene blue in carbon monoxide poisoning and have brought forward good evidence to show that it is a synergist rather than an antidote. They repeated Brooks's experiments on rats under slightly different conditions and found no beneficial effect whatsoever from methylene blue injection. In dogs they found that the methylene blue definitely hindered recovery. They attributed this action to the formation of methaemoglobin which accumulated in the blood to the extent of about 10 per cent of the total haemoglobin.

The reports of the clinical use of methylene blue in carbon monoxide asphyxia contribute little, if anything, to the problem. Five cases have been reported and in each case the methylene blue was given some time after

the poisoning when the patient was probably well on the road to recovery. All the proven cases were also given treatment with carbon dioxide and oxygen, which may have accounted for their recovery. One of Geiger's cases did not receive carbon dioxide and oxygen and made a very dramatic recovery after the injection of methylene blue, but in this case there was considerable doubt as to the cause of the poisoning. The intoxication was due to the fumes from a gas water heater which was burning a natural gas almost free from carbon monoxide.

In view of all these experiments, it seems safe to conclude that the slight improvement which has been noted following injection of methylene blue in cases of carbon monoxide poisoning is due to some non-specific stimulating action of the methylene blue, or to the effect of the injection itself. Since methylene blue has been shown to be harmful in some cases, there does not appear to be any valid reason for using it.

Among the other remedies that have recently been tried for carbon monoxide asphyxia, sulphur compounds and ultra-violet light deserve mention. Both seem to be of some value but the experimental evidence is still inconconclusive.

SUMMARY

The conclusions reached from this survey of the literature may be summarized thus:

The utility of the methaemoglobin-forming antidotes in cyanide poisoning seems to be well established. Due to its ready availability and ease of administration, amyl nitrite is the best of these antidotes for clinical use. Several of the sulphur containing compounds are of equal value. Very great antidotal action is obtained by giving the methaemoglobin-forming and the sulphur-containing compounds together.

The evidence suggests that none of the newer remedies is of much value in the treatment of carbon monoxide poisoning. Carbon dioxide and oxygen inhalation is still the best treatment for this condition.

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PUERPERAL MORTALITY: SUCCESS OR FAILURE?

IT is extremely disconcerting to be reminded that there has been no reduction in puerperal mortality in the last quarter of a century when such dramatic success appears to have attended efforts in other public health fields. Indeed, it is reported that in certain centres, as London, England, maternal mortality is increasing—mainly due to an increase in deaths from puerperal sepsis. This state of affairs is perplexing, particularly in the light of the recent careful studies conducted in New York City and in Philadelphia which have demonstrated that probably about two-thirds of all puerperal deaths are preventable in character. One is forced to consider the possibility that the work of public and private agencies, the introduction of prenatal and post-natal clinics, the educational efforts to interest women in the importance of securing medical supervision early in pregnancy, and some improvement in the teaching of obstetrics in medical schools have been of little avail. It seems especially difficult to believe that there has been no achievement when it is certain that the treatment of puerperal disease has improved in the past twenty-five years.

It may, however, be argued that there has been success and that this success has been in maintaining stationary a mortality rate which would otherwise have risen. The trend of mortality from any cause over a period of years must always be regarded with reserve because of increasing completeness and accuracy of records and it would seem that a possible explanation might be an increase in the prevalence of abortion. This would result in a larger number of puerperal deaths, since more pregnant women would die of septic abortion. There would also be fewer live births, upon which maternity risk is based. Both these factors would tend to increase the puerperal death rate as now computed. At present we have no evidence to establish such a contention.

One must either assume that failure is not real but only apparent, or else conclude that the efforts employed to date have failed. In Holland, where puerperal death rates are computed in a manner comparable generally with Canadian practice, the rate is less than two-thirds of that in Ontario. This difference is due to lower rates in each group of causes and particularly in sepsis, toxaemias and haemorrhage. This

suggests that something toward a solution might be found in a study of the methods employed in the midwifery service in that country.

There is a real need for a new attitude and a new approach to the whole question of maternal mortality. Particularly is this need evident when puerperal sepsis is considered. There is sufficient evidence to warrant regarding this disease as an epidemiological entity and to suggest that it is governed by the ordinary epidemic laws. The importance of human carriers of haemolytic streptococci who come into contact with obstetrical patients directly, or indirectly through the instruments they employ, is clearly enough defined in relation to sepsis to warrant the requirement that all those in attendance on obstetrical services be not carriers of these organisms. Nurses and house staff who are carriers should not be permitted to assume duties on the obstetrical service until laboratory examinations have shown them to be free from these organisms. Much work and inconvenience is involved in such a routine but the lives saved and the sickness prevented, as well as the valuable information gained, would be ample recompense.

Surveys such as the one reported in this issue are of great value in adding to, refreshing and clarifying our store of knowledge of the facts and factors underlying this international problem and should be continued. It is, however, impossible in covering such a large field to secure personal contact with the physician, nurse and family of the cases in question, from whom much of value might be learned. It is suggested that if financial support could be obtained a more intimate study conducted over a period of years under the direction of a medical committee might be undertaken in a large urban centre having a well-organized health service. One of the first objectives would be to define the probable proportion of puerperal deaths which are preventable in such a Canadian group of cases. Since such a survey would permit the collection of data by personal interview with all persons concerned, decisions on preventability and responsibility for death could be impartially assigned by the medical committee and a firmer basis established for progress in this field in Canada.

Puerperal death is a question of a very complex nature and no simple solution is to be expected. It is almost certain that a properly co-ordinated, carefully controlled state midwifery scheme conducted in the light of all modern epidemiological and obstetrical knowledge is essential to the solution of the problem. It is equally true that there are a number of factors the influence of which is not definitely known and on which it is extremely desirable that data be secured. Should the relative safety of delivery at home be emphasized? What are the essential factors responsible for the differences in mortality among urban and rural women? What of the social aspects? These, in the words of Sir George Newman, "are more important even than the medical issues of the maternity problem. For motherhood is not only the physical source of a people, but one of the foundations upon which a nation is built."

PUBLIC HEALTH ENGINEERING

*Water Supplies on Common Carriers**

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SUBSECTION (d) of Section 9, Part II, of the Department of Pensions and National Health Act provides for 'The supervision, as regards the public health of railways, boats, ships, and all other methods of transportation'. This also is in accordance with the definition of the jurisdiction of the Federal Government as laid down in the British North America Act.

With the above authority in the background and two Orders-in-Council dated the 19th of June, 1923, and the 25th of February, 1930, much has been accomplished along the lines of sanitation in connection with common carriers. Generally speaking, the drinking water on passenger trains and passenger vessels can be regarded as safe, as such water supplies are subject to frequent sampling and analysis. Particularly is this the case in regard to common carriers in international traffic, as the result of an agreement between the United States Public Health Service and the Department of National Health of Canada. By means of this agreement, inspection and certification of drinking and culinary water supplies used by common carriers in international traffic, as well as the supervision of drinking water supply systems on vessels operating on the Great Lakes and boundary waters, is carried out.

Water supplies in the United States used by Canadian carriers are examined by United States health authorities and certificates furnished, and similarly Canadian water supplies used by common carriers owned in the United States are checked by Canadian health authorities and certificates furnished to the persons concerned.

As the health departments of the provinces of Prince Edward Island, Nova Scotia and New Brunswick do not include public health engineers among the personnel of their respective professional staffs, arrangements have been made for co-operation with the Department of Pensions and National Health, by means of which sanitary surveys and allied public health engineering undertakings are carried on by an engineer of this service and, in return, laboratory services are freely given by the provincial health departments. A somewhat similar arrangement has been made for the province of Manitoba. In the other provinces much is done through co-operation between the provincial public health engineering service and the engineers of this service. Regular examinations are made throughout the year at points throughout Canada.

Some idea of the work involved may be obtained from the statement that in addition to the lines of the Canadian National Railway and the Canadian Pacific Railway that cross the international boundary between Canada and the United States, there are twelve other railways which regularly operate passenger services in similar international traffic. A check is kept also on the drinking and culinary water supplies of 165 lines of vessels that are regularly engaged in international freight and passenger service.

During the season of navigation of 1933, a total of 2,203 water samples

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was collected for analysis from the drinking and culinary water supplies of vessels in such traffic.

A measure of the importance of a safe water supply for drinking and culinary purposes aboard vessels is indicated by consideration of the number of passengers carried in a single season. Thus during the season of navigation of 1933 the steamships that ply into Canadian ports and whose water supplies are under the supervision of this branch, carried 11,914,465 persons, in addition to the thousands of men who are regularly employed aboard these vessels.

Similarly the railways of Canada alone carried 21,099,582 passengers in 1932, in addition to the army of employees that are engaged in the operation and maintenance of the lines.

MENTAL HYGIENE

Psychiatric Social Work with the Maladjusted Child of Normal Intelligence

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ONE of the functions of the Division of Mental Hygiene of the Department of Public Health, Toronto, is the study and supervision of a selected group of socially maladjusted children of normal intelligence. At the present time there are under supervision approximately sixty children of both sexes ranging in age from seven to fifteen years, from about twenty different schools. All possess normal intelligence, the intelligence quotients ranging from 85 to156.

Owing to the fact that there is only one person in our division doing this particular type of work, there must be a process of selection. Thus our attention has been directed towards the shy, introverted type of child, the child with fears, the oversensitive child, the child who does not experience success either in his studies, games or social contacts, the child with an invalid reaction and the child with temper tantrums. During the past year a further selection has been made by the psychiatrist in referring for supervision only those children from more promising types of homes, where the parents are intelligent and co-operative.

Our efforts have been chiefly directed towards parent education. It is amazing how helpless some intelligent parents can be when faced with a problem of child conduct. In these times of stress and economic strain and the old order so obviously changing, we have considerable sympathy with parents who have been reared under the old order, for their difficulty in facing the fact "that things are not as they used to be" is often tragic.

The children with whom we work have, as a rule, been problems over a period of years and it is only when their anti-social conduct become marked that we find them. When parents are questioned as to early signs of behaviour difficulties, and their treatment, the information supplied is usually rather inaccurate; there is always the tendency to state that the child gave no trouble until he went to school, the parents not realizing that it may have been

his poor preparation for co-operating with others and poor habits of application and conduct in his pre-school years that started the difficulties when he went to school.

Our attention is drawn to these children by the principal, teacher, parent, or public health nurse who arranges an appointment with the psychiatrist. If the psychometric test proves that the child's intelligence is normal and the psychiatrist, on interviewing either one or both parents, finds that there is need for specialized investigation and supervision, the case is referred to the Psychiatric Children's Worker.

Prior to the examination the public health nurse provides a social history; depending on various factors such as language difficulty, reticence of parents, etc., this history may be very complete or rather incomplete. The teacher sends a report of the child's ability in his various subjects, and of his attendance, whether regular or irregular; and also comments on his personality traits as they appear in the class-room and playground. These reports, together with the child's school card, showing his marks, promotions, attendance and some account of his physical condition since his entrance into school, are all studied by the psychiatrist, who then makes definite recommendations as to further investigation and treatment.

The next step is to visit the home. The first visit is usually of a lengthy nature, as one may find it necessary from time to time during the interview to listen to an account of other disturbing conditions in the home and many illuminating facts may be divulged that did not appear in the strange and new environment of the clinic. This is where patience is a virtue on the part of the visitor.

Next the school is visited. If the principal is interested, here is an excellent opportunity to enlist co-operation in the field of mental health for the child. Some information regarding the child's home conditions is given to both principal and teacher; here one must use tact and discretion.

A brief discussion of some of the different types of maladjusted children may be of interest.

There is the shy, seclusive child who finds it difficult to make friends, who cannot enter into games without feeling self-conscious. Does the lack of attractive clothing make the child feel conspicuous? Is there any physical defect present that marks him out among other children, or physical disease that prevents him entering into games? Has he been so frequently compared with a brighter child at home or in school that he feels insecure and unwanted? Or is his mental age so far ahead of his chronological age that he has little in common with his classmates? These latter children are most interesting types to study, for although possessed of intelligence that should ensure success in the generally accepted sense, they often develop into most unhappy adults due to early habits of seclusiveness, aggressiveness and consequent inability to make friends. It has been said, and quite truly I think, that only one thing is sadder than a lonely child and that is the lonely adult into which he develops.

Then we have the child who constantly repeats grades. Is his lack of skill ridiculed by those who should treat it with a sympathetic understanding? Is he deterred from effort by the very habit of failure, by the feeling that he cannot do it or is not expected to make good?

With many parents success at school means graduating from high school at the age of seventeen, or keeping up the record of an older brother or sister, or doing as well as the child across the street. We have come in contact with many children who have developed various forms of social maladjustment simply as a result of such parental pressure.

The child who, at the age of nine or ten, cannot go to sleep without a light in his room is probably the victim of an over-solicitous parent,

or, on the other hand, the type of parent who uses as punishment threats of dark rooms with unknown terrors.

Then there are those suffering from an invalid reaction as a result of much attention during an illness or of association with a neurotic adult. The plea of poor health and "nerves" is used as an excuse for avoiding irksome tasks, both at home and at school. These children often go from physician to physician, or clinic to clinic, suffering from physical symptoms with no apparent organic basis.

Another problem is the child who will not obey. Our experience with these children has brought us in contact with two types of parents: those who adopt a negative attitude and keep up a constant stream of "don'ts", and those who use their role as parents in order to become dictators.

In connection with fears, the case may be quoted of a child of seven who was brought recently to our attention with a history of night terrors, afraid to sleep in a bed by himself. While discussing the matter with the mother it was found that all the children, numbering six, had night terrors. On visiting the home one found a story of nearly three years of unemployment, the parents sensitive at accepting help from the city, the father irritable and the mother complaining of peculiar sensations in her head, sleeplessness, indigestion, no interest in her home, being impatient with the children, and a history of money losses and nervous "breakdowns" throughout the family connections.

There seemed little use trying to help this one small child while every factor in his surroundings contributed to failure. As we saw it, the first step was to give the mother a rest away from her family. With the co-operation of the family physician, the Hillcrest Convalescent Home and the Visiting Housekeepers' Association, we were able to arrange for her to remain away from home for a period of six weeks. So far as she herself is concerned there is much yet to be done; but the mental health of the children has improved 100 per cent, due to the common sense of the housekeeper and the co-operation of the father.

This case will illustrate the fact that the adult pattern in the home is of the utmost importance. How can a parent with faulty mental attitudes help her child to develop an integrated, adaptive and adjusted personality? How can a social worker, public health nurse or any other individual engaged in this field accomplish much if she herself does not possess health in its broadest sense?

Before concluding, the placing of children in an environment outside their own home, in foster homes, should be mentioned. Where we have been able to accomplish this, we have had much success; in order to do this it is necessary to enlist the aid of a satisfactory and co-operative child placing agency.

Recreation centres such as are provided by the Y.W.C.A., Y.M.C.A., Boy Scout Movement, C.G.I.T., etc., have been of great assistance to us where more recreation for the child was indicated.

The aim of our work in connection with these types of children can be adequately described in a quotation from Doctor Ira S. Wile.

"The mental hygiene approach is interested in human stability. It would promote all that would advance human balance in so far as we have knowledge of its attainment as a by-product of living. It would seek to cultivate courage and independence, friendliness and co-operation; it would foster all plans designed to encourage the domination of intelligence over emotional impulsiveness. It would recognise that man suffers because his intelligence and his emotions do not work harmoniously in the interest of his physical and social well-being."

BOOKS AND REPORTS

The Physiological Effects of Radiant Energy. *By Henry Laurens. Published by the Chemical Catalogue Company, Inc., 330 West 42nd Street, New York,* 1933. 603 *pages. Price,* $6.00.

This volume is an American Chemical Society Monograph, one of a series which contains a number of valuable reviews, and which serves the purpose of making reviews readily available, thus lightening the stupendous task of keeping up with the literature. The author is Professor of Physiology in Tulane University and is well known for his research contributions.

After a brief introduction, eighty pages are devoted to the physics of radiant energy. This chapter summarizes methods for measuring radiant energy and results which have been obtained, particularly for sunshine in various localities. Then follow fourteen chapters dealing with physiological effects attributed to radiant energy, with a final chapter on the mechanism underlying these effects.

In a book of this type it is perhaps inevitable that errors should creep in and a number could be listed. Two are selected, which to the writer emphasize the advisability of critical editing. "Ultra violet energy in sunshine activates the vitamin D in the skin" (page 285). "Centanni (1930) discovered that pregnancy is extremely susceptible to the action of ergosterol, which may constitute a grave insult . . . if the dose be excessive, and if the mother survive, the insult is expressed in one of two ways: conception is inhibited or the foetus dies" (page 473).

Naturally a major portion of the book is devoted to rickets. An emphasized opinion is best illustrated by a quotation: "The action of a foodstuff artificially rendered antirachitic by irradiation is identical with the action of a naturally occurring foodstuff containing the antirachitic factor." Accumulating evidence indicates that this is unlikely and that several distinct antirachitic factors exist, which do not give equivalent responses in various species. This has been clearly demonstrated, for example, in chickens and in humans.

One should not recommend this book to those readers who are unacquainted with the subject and who desire an easily read and brief review. To workers in this field, however, the volume should prove to be a most useful reference. One cannot leave the book without being convinced that Professor Laurens has accomplished a most arduous task in summarizing a very extensive literature.

E. W. McH.

Mental Hygiene in the Community. *By Clara Bassett, Consultant in Psychiatric Social Work, Division on Community Clinics, National Committee for Mental Hygiene, Inc. Published by the Macmillans in Canada, St. Martin's House, 70 Bond Street, Toronto,* 1934. 394 *pages. Price,* $4.20.

This book is very comprehensive and complete; in a sane and well-balanced manner it points out the relation of mental hygiene to various community problems. The thirteen chapters show how important a knowledge of mental hygiene may be in producing a healthier and happier community spirit.

The author knows her subject well and very convincingly shows the importance of mental hygiene to the physician, the lawyer, the nurse, the teacher, the social worker, and the clergyman. Its importance in industry and recreation is also discussed. The list of suggestions at the end of each chapter is of great practical value.

All who are interested in the improvement of the physical and mental health of their communities will find the book most helpful and instructive.

R.R.M.

CURRENT HEALTH LITERATURE

These brief abstracts are intended to direct attention to some articles in various journals which have been published during the preceding month. The Secretary of the Editorial Board is pleased to mail any of the journals referred to so that the abstracted article may be read in its entirety. No charge is made for this service. Prompt return (after three days) is requested in order that the journals may be available to other readers.

Outbreak of Milk Poisoning due to a Toxin-Producing Staphylococcus found in the Udders of Two Cows

This report deals with a series of outbreaks of acute food poisoning which occurred in a preparatory school. Six outbreaks occurred in a period of 7 weeks, giving rise to a total of 242 cases of poisoning in 97 persons. The illness was characterized by sudden nausea with vomiting and diarrhoea and sometimes collapse. There was no rise in temperature and recovery rapidly followed emptying of the stomach. In each outbreak all cases had their onset within 15 to 30 minutes of each other. The consistency of the onset in 3 hours following a meal suggested food as the most probable vehicle. Chemical examinations of the articles of food proved entirely negative. Bacteriological examination showed that haemolytic staphylococci were present in enormous numbers in samples of the milk and vomitus from cases. It was also established that, with one doubtful exception, no case of poisoning occurred in individuals who had not partaken of milk at the meal preceeding each outbreak.

Filtrates were prepared from cultures of the staphylococci from the milk and vomitus and these, administered to human volunteers, produced symptoms clinically identical with those occurring in the outbreaks. For a period of 5 weeks, 3 specimens per week of milk from the 13 cows of the school farm were examined. Two cows were found to be almost consistently excreting haemolytic staphylococci which produced toxic filtrates. The milk from one of these averaged about 4,500 staphylococci per cc., the other about 2,900 staphylococci per cc. From a high count the number of organisms decreased in successive samples until few or none were demonstrable and this would be followed by a sudden and tremendous increase. This cyclic excretion suggests the presence in the cows' udders of pus pockets which intermittently discharged their contents into the milk.

The presence of the staphylococci in the milk did not, alone, explain the outbreaks. The households of the principal and the farm manager escaped, apparently because their milk was rapidly chilled and efficiently refrigerated. The supply for the school was stored in large cans and kept at a higher temperature. This indicates formation of the toxin as a result of multiplication of the staphylococci in the milk.

James A. Crabtree and William Litterer, Am. J. Pub. Health, *24*: 1116 (Nov.), 1934.

Intestinal Tuberculosis in 1,400 Autopsies

In a review of 1,400 autopsies, more than two-thirds of the cases of fatal phthsis had ulcerative tuberculosis lesions of the intestines, only one-tenth of the ulcers in the series being of classical type. Ulcerative intestinal tuberculosis was found as a complication of tuberculous pulmonary disease in over four-fifths of the coloured but only two-fifths of the white group.

P. M. Crawford and H. P. Sawyer, Am. Rev. Tuberc., *30*: 568, 1934.

Psychological Considerations involved in the Application of Motor Driving Tests

Capability for driving depends upon a number of fundamental considerations and the motor driving tests which have been devised are designed to assay these, viz., the ability of the person tested to meet hazards, and to judge accurately the speed of moving objects and estimate spatial relationships. One of the most important defects, slow reaction time, is extremely dangerous and regardless of knowledge of regulations and road manners, such an individual must sooner or later incur road accident. Other advantages of motor driving tests are (1) reduction in training time of beginners, (2) reduced running costs and (3) acquainting persons with a knowledge of their weaknesses.

G. H. Miles. Human Factor. *8(2)*: 407 (Nov.), 1934.

Studies in the Variability of Tubercle Bacilli

Of five cultures of B.C.G., when received, one showed a few S type colonies. The other four consisted only of R type. After a long series of cultures, S type colonies were isolated from two of the latter strains, but no stable S types could be obtained from the other two. By means of complement fixation reactions it was shown that animals immunized with S organisms develop a specific S antibody which is not found in the serum of animals treated with R organisms or the ordinary form of B.C.G. From this the authors infer that a pure R strain is not likely to produce an effective immunity.

G. B. Reed, J. H. Orr and C. E. Rice, Canad. J. Research, *11*: 362 (Sept.), 1934.

Antituberculosis Vaccination

The writer discusses immunization against tuberculosis by the injection of (1) living virulent tubercle bacilli, (2) heat-killed tubercle bacilli, and (3) living avirulent tubercle bacilli (B.C.G.), and states that he is now definitely opposed to "tuberculization".

S. A. Petroff, New Eng. J. Med., *211*: 677 (Oct. 11), 1934.

Sustaining Membership

Pursuant to the changes in the By-laws of the Association establishing the basis of sustaining membership, the Executive Committee has pleasure in announcing the acceptance of this form of membership by

The
London Life Insurance Company

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Manufacturers Life Insurance Company

The
Canada Life Assurance Company

Through the adoption of this form of membership by various institutions and companies, the Association is enabled to extend its activities in the advancement of preventive medicine.

CANADIAN
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One day in the life of a

Visiting Nurse

SHE is but one of many women whose days are too short to do all they are asked to do and indeed eager to do, in accordance with the doctor's orders, for those who need their skilful and sympathetic care and direction in hygiene.

Looking for no praise, this Visiting Nurse turned in her report for a single day. From early morning until late afternoon every minute was occupied. But there was no place in her record for her own energy, tact, courage and resourcefulness, or for fatigue, climbing dark stairs, constant drain on sympathy for acute suffering or lost hope.

Her appointments, which averaged about an hour each, began with a call on Mrs. Smith—an enema for intestinal disturbance, as ordered by doctor. Then Tim Kelly—lobar pneumonia. Next, Mrs. Jones and new baby. After her, Jean Baptiste—an infected leg. Audrey Robinson next—under doctor's orders, gave insulin injection for diabetes. Mrs. Marziotti—prenatal care. Mr. Simmons—a chronic invalid: paralysis. Finally, Lucy Carleton — diphtheria: assisted the doctor in immunizing the other children; arranged home for communicable disease isolation.

The Visiting Nurse Service is one of North America's distinguished contributions to the health movement of the world and has been adopted in other countries. Here it is supported by patients whose payments are supplemented by those of organizations that recognize the vast importance of this work which includes education in health. The Visiting Nurse wholeheartedly extends to each patient the benefit of her expert training.

The wage-earner who cannot stay at home when there is illness in the family, but who can afford the part-time service of a Visiting Nurse, goes to work with a lighter heart knowing that she will call at a definite time to do what is required. When possible the Visiting Nurse teaches some member of the family how to give bedside care before she hurries on to her next patient.

Through your telephone book or your doctor, you can find out whether or not there is a V.O.N. or other Visiting Nurse Service in your neighborhood. These trained graduate nurses are on call in more than 80 cities and towns in Canada. The bedside care given by them may help turn a serious illness to full recovery of health and strength.

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